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a record of research

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SEPTEMBER, 1929

THE SPREAD OF GENERATIONS

BY ALFRED J. LOTKA

Where inheritance is regulated in accordance with the right of primogeniture, this selection operates to mark out a line of successive male generations of minimum duration—always from father to eldest surviving son. So far as I am aware, no official stamp has ever been placed upon a similar chain of generations traced always through the youngest surviving son. But there is no logical obstacle to our fixing our attention upon such a chain. We then see two perpetually diverging lines of descent, representing the two extremes between which successive generations of males are comprised-in-time. Evidently, a similar scheme can be developed for the female line of descent, from mother to daughter throughout; and though this lacks the legal background of the male line of descent, it is, from the standpoint of biology and of population statistics, if anything, of even greater interest than the corresponding scheme of the male line.

A diagram will help to illustrate the matter, and will at the same time serve to bring into prominence questions that naturally arise.

In Figure 1 the horizontal scale is a time scale of years. This scale is repeated at successive equal vertical intervals of 28.5 years, this being the mean length of one generation, reckoned from mother to daughter, in the United States, according to the statistics of the last census year, 1920. These successive horizontal scales are numbered on the left with the ordinal numerals 1, 2, . . . which denote that the scales so numbered have been assigned to the first, second, generation, etc., respectively. The horizontal line at the top would similarly correspond to the zero generation.

The zero generation here contemplated is a batch or cohort of say 100,000 females all born on one day of the year zero, which, to give the matter a more concrete touch, has been designated as the year 1900. Their daughters are the first generation, their grand-daughters the second, and so on. Let us now consider over what portions of the successive time scales these generations will extend. Since the zero generation was all born on one day in the year zero, its members will all be ten years old at the same time, namely in the year ten(or 1910 on the scale of dates). About this time, that is, some time between their tenth and the fifteenth year, they will begin to reproduce, and they

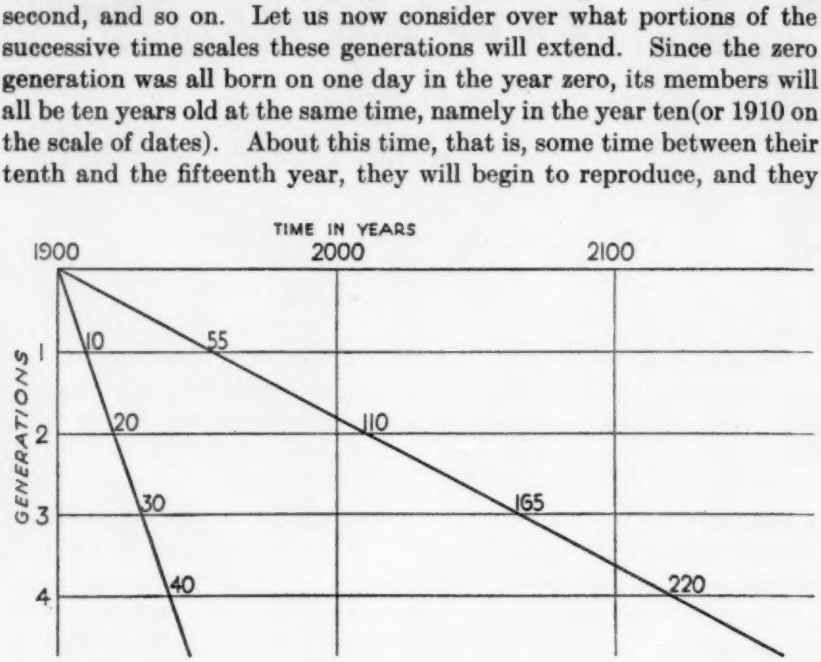

FIG. 1.

will continue to do so until they are about fifty-five years old, that is, in the year fifty-five (1955). Thus the first generation will extend over a stretch from somewhere about ten to fifty-five on the time scale marked 1. (In the United States during the years 1917 to 1924, there occurred a total of over 5,000 births by mothers under fifteen years of age. The round figure ten will here be used, for convenience, as the extreme lower limit for the female reproductive period. The exact location of this limit is of no consequence for our argument, as will be seen in what follows.)

When some of the members of the first generation are themselves ten years old, they in turn will begin to reproduce. This will first occur when the oldest of them are ten years old. But the oldest of them were born in the year ten; hence the reproduction of the first

generation, and the births in the second generation, will begin in the year twenty (1920). They will continue until the youngest of the first generation are fifty-five years old. But the youngest of the first generation were born in the year fifty-five. Hence reproduction in the first generation and births in the second, will extend to the year 110 (dated 2010). In the same way it is easily seen that the births in the third generation extend from the year thirty to the year 165, and so on. Evidently, we can mark off a triangular field, as shown, in our diagram, which contains the births in all the successive generations. Suppose, now, that we draw a vertical line through the year 2100 on the time

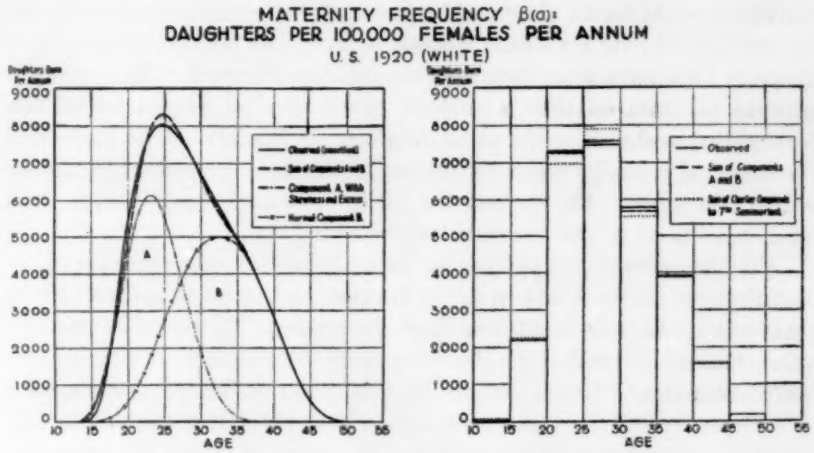

FIG. 2.

scale. This cuts through the base lines of the fourth to the twentieth generation. From this we should conclude that in the year 2100 there will be living, as contemporaries, members of the fourth to the twentieth generations (the latter beyond the limits of Fig. 1). We shall see presently that this conclusion must be modified materially in its quantitative aspect; but qualitatively it gives us a conception of the overlapping of generations. It is rather curious to reflect that living side by side with us may be persons who, as regards order of generation, stand to us in the relation of great-great-great-great-grand uncle or aunt for example, if we may be allowed to use the words uncle, aunt, in a somewhat extended sense. But let us proceed a little more cautiously now. Our diagram shows that *some* persons of such remote

generations would coexist with us. But *how many?* We have fixed extreme limits between which the successive generations must be comprised; but how are they distributed over these successive ranges? For the first generation the answer comes readily. These are the daughters of mothers of the zero generation who, themselves, were all born at the same time, namely $t = 0$, and who are therefore each of age t at time t. The curve representing the time distribution of births in the first generation is therefore simply the frequency distribution of births (daughters) classified according to the age of the mother, and corrected to include only those mothers of the zero generation who survive to the several ages of the reproductive period. The frequency distributions of births classified by the age of the mother are published by the United States Census Bureau in five year groups, so that their graph is a histogram, as shown on the right in Figure 2. By a suitable process of interpolation a smooth curve can be drawn to fit this histogram, as shown in the same diagram, on the left. The correction for survival is easily made by means of the data contained in the so-called life table. The figures for 1920, the last federal census year, have been used in the example here shown.

For the second and the higher generations the construction of the distribution curve is not quite so simple, and it must suffice here to state results without exhibiting their derivation. It can be shown that the standard deviation of the frequency distribution of the second generation is $\sqrt{2}$ times that of the first; that of the nth generation is $\sqrt{n}$ times that of the first; and a similar simple relation connects all the other characteristics (seminvariants) by means of which the frequency distributions of successive generations are defined. One result of this particular form, which the relation between successive generations takes, is that the frequency distribution of higher generations approaches more and more nearly to the normal, even though that for the first generation in actual fact departs very materially from the normal. This approach toward the normal distribution in successive generations is graphically exhibited in Figure 3, which is so drawn that a normal distribution appears as a straight line, the tangent of whose slope is numerically equal to the square of the corresponding standard deviation, i.e. to the ordinal number of the generation in question. It will be seen that the points (black) representing the actual distribution in the first generation are rather widely scattered about a line of unit slope (that is, 45 degrees to the horizontal). Those for the second generation are more closely clustered about a line of slope 2; and those

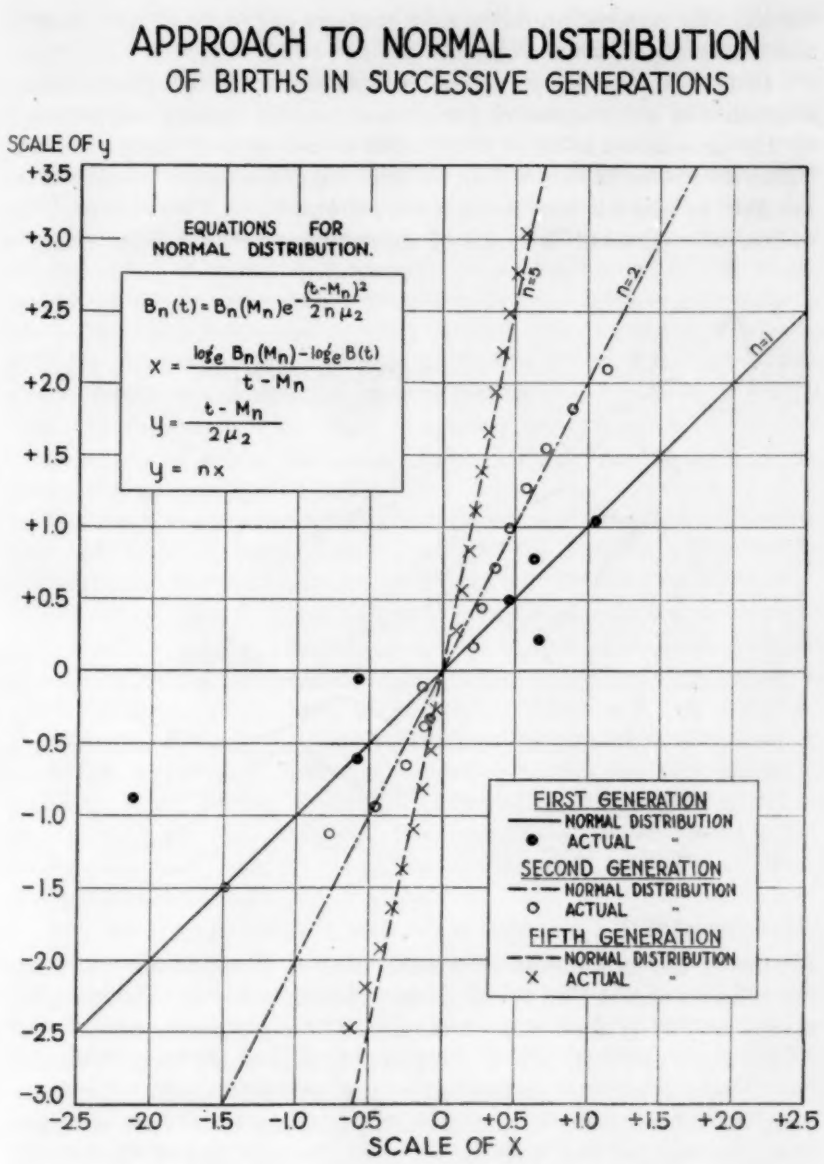
APPROACH TO NORMAL DISTRIBUTION
OF BIRTHS IN SUCCESSIVE GENERATIONS
SCALE OF y
+3.5
+3.0
+2.5
+2.0
+1.5
+1.0
+0.5
0
-0.5
-1.0
-1.5
-2.0
-2.5
-3.0
EQUATIONS FOR NORMAL DISTRIBUTION.
$B_n(t) = B_n(M_n)e^{-\frac{(t-M_n)^2}{2n\mu_2}}$
$x = \frac{\log_e B_n(M_n) - \log_e B(t)}{t - M_n}$
$y = \frac{t - M_n}{2\mu_2}$
$y = nx$
n=5
n=2
n=1
FIRST GENERATION
NORMAL DISTRIBUTION
ACTUAL
SECOND GENERATION
NORMAL DISTRIBUTION
ACTUAL
FIFTH GENERATION
NORMAL DISTRIBUTION
ACTUAL
-2.5 -2.0 -1.5 -1.0 -0.5 0 +0.5 +1.0 +1.5 +2.0 +2.5
SCALE OF X

FIG. 3.

for the fifth generation (shown by crosses) are quite closely arrayed about a straight line of slope 5.

Instead of quoting numerical values of the higher characteristic constants of the successive generations, it will be more appropriate on the present occasion to exhibit the actual curves, drawn to scale. These are shown in Figure 4 for the first ten generations. And now we are able to make a number of observations which a mere knowledge of the extreme possible range of successive generations, as shown in

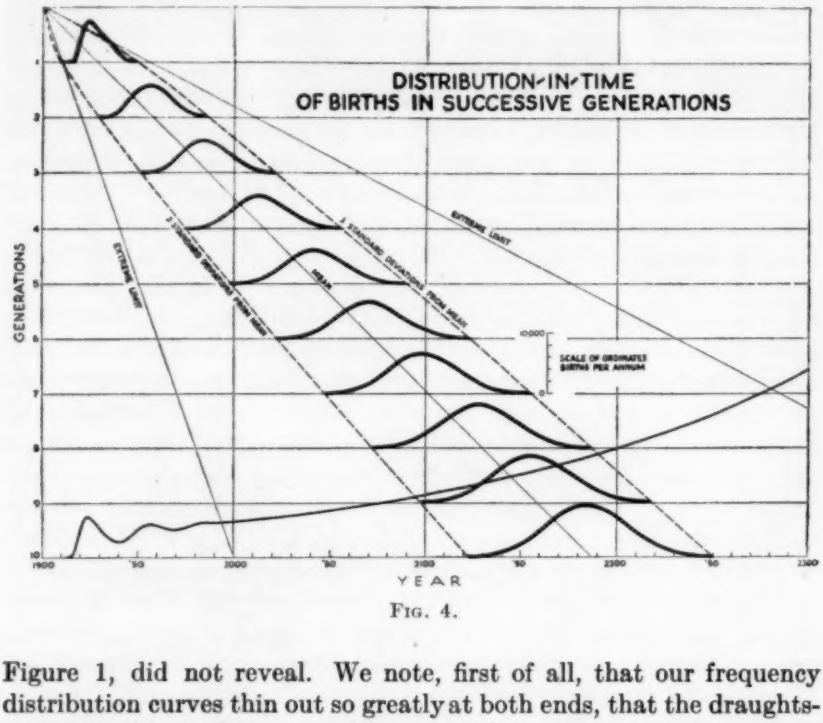

FIG. 4.

Figure 1, did not reveal. We note, first of all, that our frequency distribution curves thin out so greatly at both ends, that the draughtsman is unable to show any visible area of the frequency curves at all on the right and left, over a considerable part of the range which we had at first (Figure 1) staked out as the area within which births are comprised, for successive generations. As a matter of fact, of course, it will be recalled that well over ninety-nine per cent of all the cases included in a normal frequency curve are comprised within three standard deviations to the right and left of the mean, and this remains true for most unimodal distributions, even if they depart materially

from the normal. In the diagram Figure 4, in addition to the extreme range—the triangular area within which all *possible* births are comprised—there is also shown a more restricted area bounded on the right and left by curved lines, which have been spaced at each generation three standard deviations on either side the mean. This area comprises *practically* all the births. Thus the actual range falls short of the extreme range staked out as a triangular area in Figure 1. This fact is also obvious when viewed from another angle. About one female in ten thousand per annum, under the age of fifteen years, becomes a mother of a daughter in the United States. But cases in which there is a succession running through two or more generations, in which mother, daughter, granddaughter, etc., are each born by her mother at the age of fifteen or under, must be so scarce as to be practically non-existent. This, then, is another interpretation of the fact that the area of actual births falls short of the triangular area of the extreme possible range of births.

We are now prepared to look a little more closely into a question that was touched upon above: namely, the number of coexisting generations. We shall have to revise our first estimate in the light of what has just been said: namely, that *actual* births do not occur right up to the limits of the triangular area. If we look again at the vertical line drawn through the year 2100 on the time scale (Fig. 4) and observe, this time, through how many of the frequency curves it cuts *within the area bounded by the two curves that measure three standard deviations on either side the mean*, we find that, according to this way of reading the diagram, only the sixth, seventh, eighth and ninth generations will be reproducing at this time. A few possible stragglers from other generations (e.g., the fifth and the tenth) may be disregarded as negligible in the total.

But this is an incomplete view of the situation. It is true that the generations mentioned will all be reproducing at the time $t = 200$ (date 2100). But their relative contributions to the total birthrate will be very unequal. Evidently the seventh generation will be contributing most, and the ninth very little. In fact, the contributions of the four generations, (sixth, seventh, eighth and ninth) will be as shown in Table 1, the figures being computed on the basis that the initial births in the zero generation were 100,000. It will be seen that two hundred years after the birth of the zero generation, the seventh generation contributes 62 per cent of the total birthrate, the sixth only 12.5 per cent, and the ninth only 1.1 per cent. A little

later, of course, the ninth generation will be contributing more and the sixth less, a gradual shifting to later generations taking place.

TABLE 1

Contribution of sixth to ninth generations, to total birthrate, two hundred years after birth of zero generation

GENERATION	BIRTHS PER ANNUM					
	Computed Directly		Computed By "Skew" Frequency[1]		Computed by "Normal" Frequency[2]	
	Number	Per cent	Number	Per cent	Number	Per cent
Sixth	1,307	12.5	1,275	12.2	1,480	14.2
Seventh	6,475	62.1	6,567	62.6	6,555	63.1
Eighth	2,533	24.3	2,481	23.7	2,284	22.0
Ninth	113	1.1	164	1.6	71	.7
	10,428	100.0	10,487	100.1	10,390	100.0

[1] $\dfrac{N_0 R_0^{n+j}}{\sqrt{2\pi(n+j)\mu_2}}\, e^{-\frac{j^2\mu_1^2}{2(n+j)\mu_2}}$

[2] $\dfrac{N_0 R_0^{(n+i)}}{\sqrt{2\pi n\mu_2}}\, e^{-\frac{j^2\mu_1^2}{2n\mu_2}} = \dfrac{N_0 R_0^{\left(n+\frac{\sigma^2 l}{2}\right)}}{\mu_1\sigma\sqrt{2\pi}}\, e^{-\frac{1}{2\sigma^2}(j-\sigma^2 l)^2}$

where N_0 = number of births in zero generation = 100,000

R_0 = ratio of total births in two successive generations = 1.166

$n = \dfrac{t}{\mu_1}$ = ordinal number of generation about which distribution is reckoned = $\dfrac{200}{28.47} = 7.025$

$n + j$ = ordinal number of particular generation whose contribution is sought

μ_1 = mean of frequency function $s(a)m(a)$, and

$\sqrt{\mu_2}$ = standard deviation of $s(a)m(a)$ = 45.39

l = mean length of generations = 28.35 years.

The table also shows two columns of figures *computed* according to two formulae designated respectively as "Skew" and "Normal," the derivation of which must here be omitted. Only this explanation may be given in passing, that both formulae represent approximations to which the actual figures will approach more and more nearly as we pass to later and later generations. It will be seen that even at the time of the seventh generation the "skew" formula represents quite

closely the actual figures. The interest of the second formula resides chiefly in the fact that it is the familiar *normal* frequency distribution. For high generations the two formulae become essentially coincidental; that is to say, as time goes on, the contributions of coexisting generations approach the *normal* or Gaussian law. This is shown graphically in Figure 5, in which the solid curve represents the frequency curve

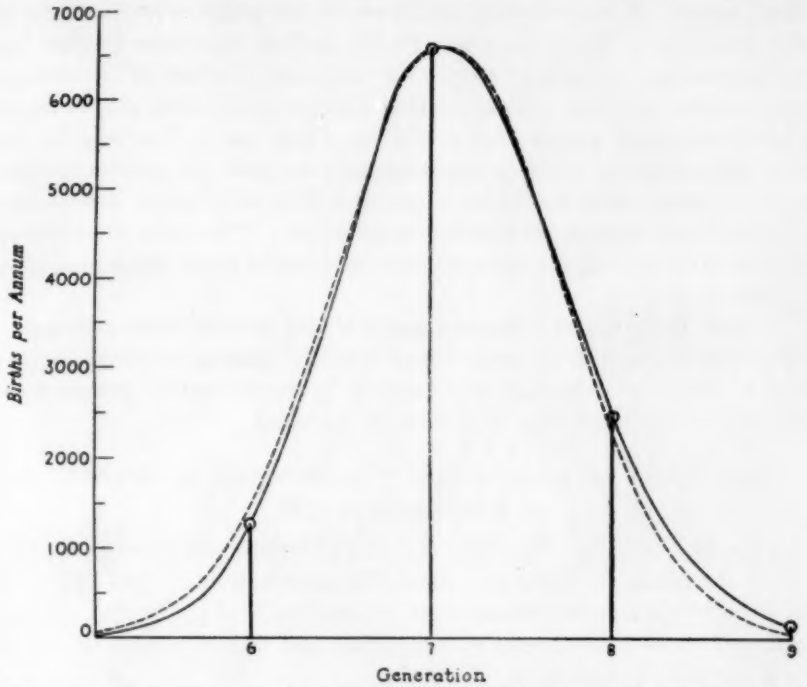

FIG. 5. Frequency curve showing contributions of sixth, seventh, eighth and ninth generations to total birth rate, 200 years after birth of zero generation. The solid curve corresponds to the "skew" formula (see Table I), the broken curve to the "normal." Only ordinates corresponding to integral values of abscissae have any concrete meaning.

defined by the more exact skew formula for $t = 200$ years (date 2100), and the broken line represents the frequency curve defined by the normal or Gaussian law for the same epoch. It is, of course, understood that for the present problem only those ordinates of these distribution curves have a concrete meaning, for which $(n+j)$, the ordinal number of a contributing generation, has an integral value. The ordinates for the values $(n+j) = 6, 7, 8, 9$ are drawn in the diagram.

The same relations are exhibited, perhaps still more plainly, in the diagram Figure 6, which represents the various relations in the form of a solid model. The frequency curves for the births in successive generations are represented as rising vertically from a floor plane, each curve being drawn in a plane parallel to the "back wall," the successive curves being spaced 28.5 years (the mean length of a generation) apart. A cross section is drawn in the plane corresponding to the instant $t = 200$ years (date 2100), and at this cross section the corresponding frequency curve for the contribution of successive generations has been drawn, cutting the planes of the sixth, seventh, eighth and ninth generations as shown. This curve, like that for the seventh generation itself, is approximately normal, and closer approach to the normal form would be found as higher and higher generations and later and later cross sections were taken. (The scale of ordinates in this drawing has, for convenience, been made more condensed than in Figure 5.)

From Table 1 and Figures 4 and 6 it will be seen that, although a considerable number of generations may be existing together, only a few of these are actually represented in considerable proportions; the remainder contribute but little to the total.

ALTERNATIVE INTERPRETATION: THE BIRTH-RATE CONSIDERED AS A SERIES OF WAVES

We have, so far, regarded the total births at any point as built up of the births in successive coexisting generations. Thus the curve of total births appears as the sum, or resultant, of a series of overlapping waves of successively wider spread and flatter profile, as shown in Figures 4, 6, and 8.

But there is an alternative way of regarding the matter. Without paying attention at all to separate generations, we can say that the birth-rate B_t at any particular instant t is the product of the number of persons of age a, times the rate of reproduction $m(a)$ characteristic of age a, this product formed for all ages within the reproductive period, and totaled for all those ages. Now the persons who, at time t, are a years old, were born at time $(t - a)$, when the number of births per annum was B_{t-a}. Of these a certain fraction $s(a)B_{t-a}$ survive to age a. Thus the sum of the cross-products mentioned above will be

$$\sum_{a=15}^{a=55} B_{t-a}\, s(a)m(a) \qquad (1)$$

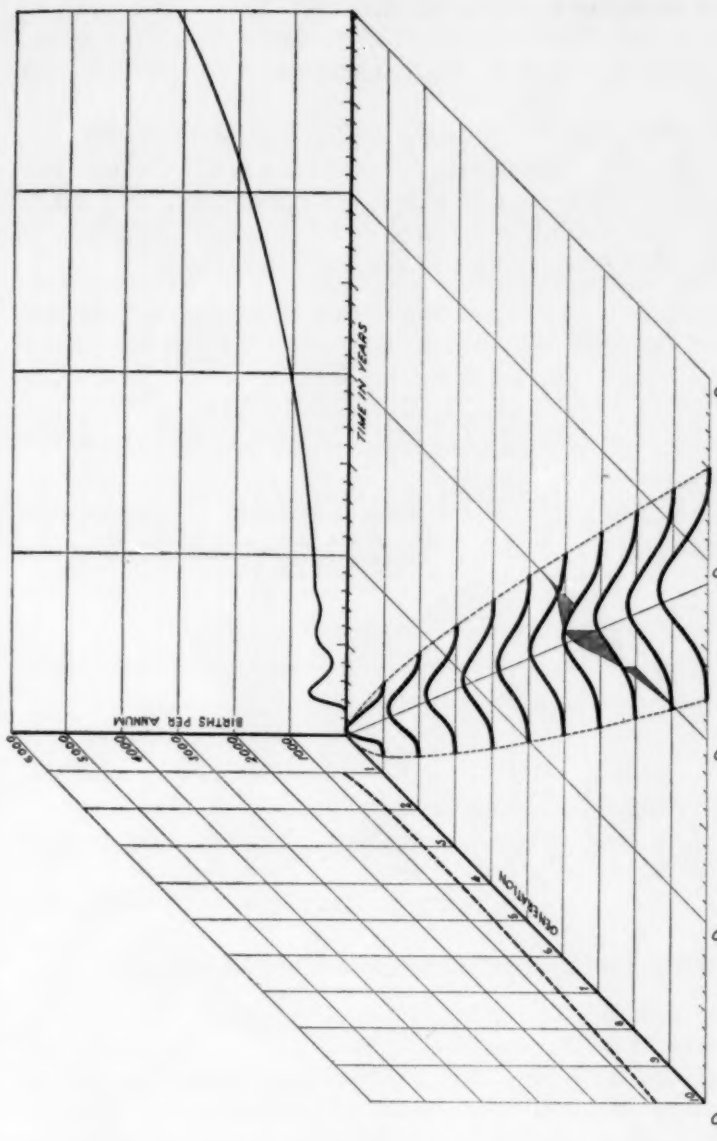

FIG. 6. Frequency distribution of births in successive generations, represented in three dimensional construction. The curve in the rear is the resultant, giving the total births ffor all coexisting generations. The curve of dashes on the left is the projection so the locus of the maxima of the individual frequency curves. Readings on the verticals cale of *Births per annum* should be multiplied by ten.

or, treating the *age* as a continuous variable

$$B_t = \int_{15}^{55} B_{t-a}\, s(a) m(a) da \tag{2}$$

This is an integral equation which defines the total birth-rate as a function of the time. For the sake of those readers who are interested in the mathematical treatment of biological problems I may briefly indicate at least the main features of its solution. This may be obtained simply enough by making the trial substitution,

$$B_t = Ke^{rt} \tag{3}$$

so that

$$B_{t-a} = Ke^{r(t-a)} = Ke^{rt-ra} \tag{4}$$

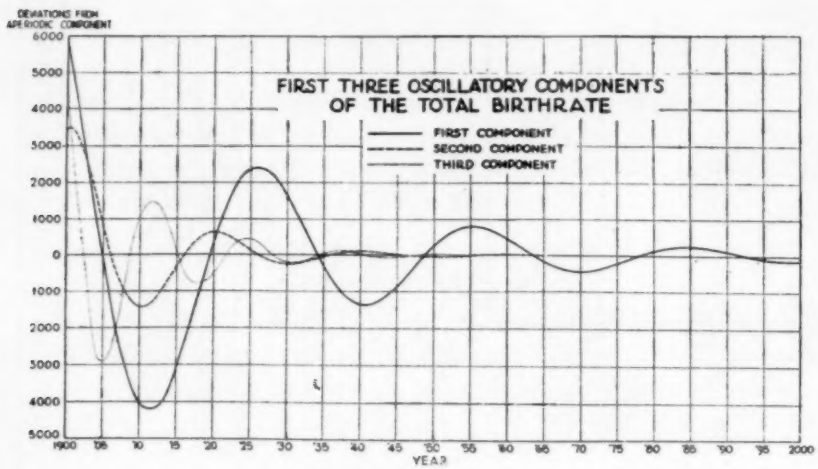

FIG. 7.

Introducing this in the integral equation we find readily

$$Ke^{rt} = \int_{15}^{55} Ke^{rt-ra}\, s(a) m(a) da \tag{5}$$

or

$$1 = \int_{15}^{55} e^{-ra}\, s(a) m(a) da \tag{6}$$

from which it is seen that $B_t = Ke^{rt}$ satisfies the integral equation (2) provided that r satisfies the equation (6), or, as we say, provided

that r is a root of the equation (6). Furthermore, it is easily seen that if r_1, r_2 . . . are several roots of (6), then a solution of (2) is

$$B_t = K_1 e^{r_1 t} + K_2 e^{r_2 t} + \cdots \tag{7}$$

It can be shown that there is only one real root, r_0, and an infinite number of complex roots. These latter contribute oscillatory or wave components to the solution, since $e^{(u + iv)t} = e^{ut}(\cos vt + i \sin vt)$. The computation of the course of the birth-rate by this method has been carried out on the basis of the same statistical material as we have made use of above. It was found that one aperiodic and the first three oscillatory components sufficed to give a very close approach to the curve of births computed by summing up successive generations.

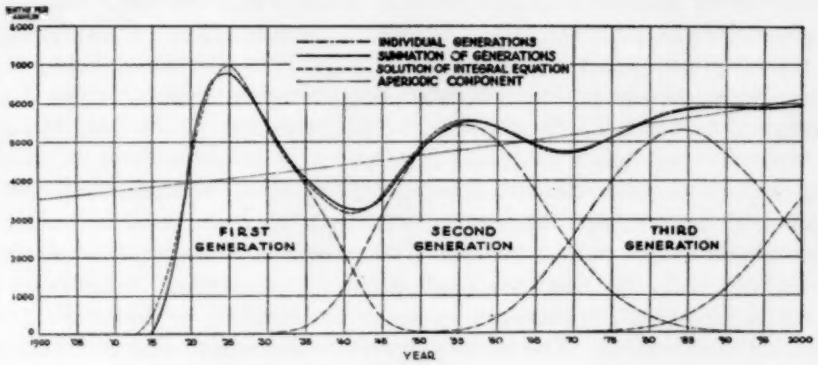

FIG. 8.

The three oscillatory components are shown in Figure 7, and their resultant, after adding the aperiodic component, is shown in Figure 8, ———— together with the curve (full line) obtained by summing coexisting successive generations of births.

A DIGRESSION INTO INDUSTRIAL APPLICATIONS

The fundamental life functions, so far as voluntary actions of the organism are concerned, are fairly summarized as the quest of food and mate, on the one hand, and the battle—be it fight or flight—with untoward influences, on the other. The organism must eat and propagate, and guard against being eaten or otherwise destroyed. These are the ends to which living organization is shaped; but in the case of man the organization extends far beyond the mere provision of bodily organs. Our whole industrial system is one greatly magnified

organization of artificial sense and motor organs and reproductive apparatus, without which it would be utterly impossible to maintain the life of the world's present human population. While these "artificial" organs differ more or less markedly from our natural organs in their mode of origin, and rather less in their characteristic structure, in *function* they fulfil identical rôles with our native organs of sense, motion and reproduction. (That machinery reproduces itself is a fact that seems to be rather commonly overlooked in text-book biology, though it must have been painfully apparent to Robinson Crusoe. In the present state of civilization we can truthfully say: *machina omnis ex machina.*)

There is one difference between our artificial aids to industry and our native equipment serving similar ends, which results in certain peculiar consequences. The duration of the natural body, as a going concern, has certain rather definite limits. These are not shared by our artificial equipment. A microscope may be antiquated in 200 years time, but it is still operative. An automobile, on the other hand, has an average operative duration of about seven years only. Out of this disparity between the duration of life of the natural body and that of its artificial accessories a variety of social and industrial problems arise. Where the life of the accessory exceeds that of the natural body, we run into legal problems to regulate inheritance; where the reverse is the case, we have the industrial problem of regulating *replacement*. The biometric aspect of the problems of inheritance of property is an inviting subject for reflection, but cannot be dwelt on here. It is intended to indicate briefly a few points of interest in connection with the bearing of our present discussion upon industrial problems of replacement and allied matters.

We will note, first of all, the mere fact that in the conduct of industrial affairs problems arise that are very similar in general character to the main problem from which we started out, the problem of the progeny of a population element. This is most easily seen by considering an ideally simple case. Let us suppose that on January 1, 1900 a commercial enterprise is started, and that on this date a lot of 1,000 auto trucks is purchased for its operative equipment. In the course of operations this original stock (the zero generation) presently begins to give out, through accidents or wear and tear. We might plot a curve showing the number of trucks scrapped as time goes on, a curve of deaths, as it were. If the enterprise is conducted at a constant level, neither growing nor contracting, each car scrapped must be replaced

by a new car, so that in this case the curve of death of the zero generation is also the birth curve of the "first" generation, and so on. If the enterprise is growing, the situation is somewhat more complicated in detail, but essentially similar in general character. In any case we shall have a succession of generations of trucks, and at any future time there will be coexisting a number of successive generations. In the first few years replacements will show more or less marked undulations, every seventh year, say, calling for specially heavy replacement budgets. But as time progresses the overlapping of successive waves causes these fluctuations to become less noticeable, and, finally, quite unobservable. From the point of view of practical operation this is, of course, a favorable circumstance.

But there is another aspect of the comparison between the method or plan of "replacement" characteristic of the organic life cycle, on the one hand, and the usual plan followed in the replacement of industrial equipment. The plan followed by the living organism is a highly ingenious one, to speak in anthropomorphic idiom. It is the plan or method of the time fuse, which, in human artefacts, is a very recent innovation. When the germ of living matter, in any of its forms, is introduced into a suitably conditioned system containing the substances needed for organic growth, this germ does not proceed to grow indiscriminately, absorbing into itself all the available substance of the system. Such conduct would lead to a condition which we, in human affairs, are wont to speak of as the exhaustion of natural resources. What actually takes place is that one part of each germ grows into a mature organism, while another part (the gonad) is placed in reserve, and provided with a time-fuse so set, that about the time when the maternal organism advances towards the termination of its career, the complementary gonad pulls the trigger that releases the time fuse, and the portion of the organic substance that had been held dormant in reserve now begins to grow. In consequence, organic growth, instead of proceeding explosively to the incorporation of all available substance into itself, proceeds at a measured pace, death keeping step, approximately, with birth, so that the material substance of the living world is kept in circulation, and continued life becomes possible, where otherwise we could expect only sporadic bursts followed by periods of exhaustion.

Nature has here shown a wisdom beyond human wisdom. For *we* have scarcely, as yet, learnt thus to regulate consumption to keep pace with regeneration. Only in very recent times has thought

been given to the problem of the exhaustion of natural resources. In one direction, it is true, great success has been achieved: the leak in the circulation of organic nitrogen is rapidly being compensated by processes of "artificial" fixation. So much so, that some prophets are already warning us of overproduction in this field. But in almost all other respects we are continuing to consume our capital of natural resources, with little thought to the problem of its replacement, if indeed, replacement is possible at all on any scale that would be of material benefit. But the optimist has no difficulty in finding a basis of fact for his cheerful disregard of omens. For if we have been burning up our *material* supply of coal, the flare in which it has gone to the sky, has so lighted up our intellectual horizon that men have become like gods; nothing seems any longer impossible, and we press on, confident that, whenever we come to a bridge, we shall know how to cross.

THE INHERITANCE OF PIGMENTATION IN THE AMERICAN NEGRO

BY IRENE BARNES

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INTRODUCTION

How is the pigmentation of the American Negro inherited? How does it behave in race crosses? Are "pure" Negro children born to mulatto parents who themselves either could or do pass as "white"? Unverified generalizations and common sense observations concerning the heredity of skin color in Negro-White crosses indicate the popular interest in the subject. Newspaper stories of the birth of a "coal-black" child to white parents, one of whom has a Spanish ancestor several generations back, are prevalent. Novelists of a certain type picture the tragic Nemesis awaiting the red-haired blue-eyed girl, the granddaughter of an octoroon, if she with her "tainted blood" marries her white lover. The modern prophet of calamity observes with foreboding that so-called scientific biology visits the "sins of the fathers" far beyond the Biblical three generations.

Popular opinion, especially in those countries in which intermixture between the Negro and any other racial group is prevalent, assumes that the result of the cross is a blend in pigmentation. Quite elaborate theories have been worked out applying the prevailing color nomenclature to the type of blend produced by specific matings.[1] Biologists in general, enthusiastic over the results achieved in utilizing the Mendelian theory as a basis of prediction in inheritance, and over its ever-extending range of applicability in the field of life below man, assume on a theory of the uniformity and universality of natural mechanisms that it probably holds in human traits. They point out that many so-called blends appear on further analysis to be explicable on the hypothesis of quantitative Mendelism as produced

[1] Pearson, Karl. Note on the Skin Color of the Crosses between Negro and White. *Biometrika*, vol. vi (1908–9), p. 348–353.

by the interaction of various numbers of multiple factors, and that there is no single well-authenticated case of blending without later segregation of traits in the descendants.[2] However, neither common sense generalizations that Negroes produce in the main Negro children intermediate between the color of the parents, nor analogical reasoning from the Mendelian mechanisms operative in the plant and animal life other than man, afford scientific proof of the nature of the inheritance of pigmentation in the American Negro.

TECHNIQUE OF PIGMENTATION MEASUREMENT

Any scientific study of the inheritance of pigmentation was almost impracticable until a method was devised to secure relatively precise quantitative expressions of the almost infinite variety of shades of pigmentation which any population, either Negroid or otherwise, presents. If one were dealing with control material and could secure a sufficient number of generations and the requisite type of crosses, a relatively small number of cases might suffice, but in dealing with human material the main reliance must be placed upon statistical analysis of large numbers of cases. The color top gives a method at once quantitative, relatively accurate, and easily applied in the minimum of time and under the various and often unfavorable situations in which biometric measurements must be taken in the field.[3] This device, now technically known as "The Pupil's Color Top," no. 8109, originally designed as a toy by the Milton Bradley Company of Springfield, Massachusetts, was first used to measure pigmentation by C. B. Davenport.[4] Mechanically it is a very simple device. Paper discs of various colors are fastened by a small wooden catch to the round flat base of a child's toy top, giving a surface which presents segments of discs occupying varying proportions of the field. Of the color discs which come with the top, Davenport and most later workers have used the black, ox-blood red, yellow, and white in matching the pigmentation of the Negro. The black and white discs are identical with the standards in Ridgway;[5] the yellow is a pure spectrum yellow, identical with Ridgway's Lemon Yellow; while the red is midway

[2] Walters, H. E. Genetics. *New York*, The Macmillan Co., 1919. p. 181–182.

[3] Davenport, G. C. and C. B. Heredity of Skin Pigmentation in Man. *American Naturalist*, vol. xliv, p. 642–672.

[4] Davenport, C. B. Heredity of Skin Color in Negro-White Crosses. Carnegie Institution of Washington, Publication No. 188, 1913.

[5] Ridgway, R. Color Standards and Color Nomenclature. *Washington*, 1912.

between Carmine and Ox-blood, tone *j*, which is quite standardized by the Bradley Company with 41 per cent spectrum red and 59 per cent black.[6] Thus every unit of red on the spinning top represents approximately 41 per cent of a unit of spectrum red and 59 per cent of a unit of black which in reality belongs to the black sector.

The procedure in using the color top to measure pigmentation has been described by Davenport,[8] Todd,[9] and Herskovits.[10] The circumference of the cardboard base of the top is divided into twenty units, each representing 5 per cent of the total circumference of the circle. When discs of the four colors described above are fastened to the top, percentage readings of the proportion of the circumference occupied by each particular color may be taken. The discs are manipulated with forceps, and changed after every ten or twelve readings, as the slight amount of grime on the edge of the segments may make a difference of as much as 10 per cent in the readings.[11] By varying the proportions of the total surface of the top occupied by the sectors of black, red, white, and yellow, and whirling the top, a blend may be produced to match the pigmentation of any individual.

In the collection of the data of this study, the following technique was employed. The skin of the upper outer arm was used, as it is easily accessible, and less influenced by either weather or cosmetics than any other part of the body. The individual whose pigmentation was to be measured rested his arm upon a white paper which had been placed on a flat surface, while the upper arm was covered with a white paper in which a small hole had been cut. This exposed a portion of skin surrounded by white for measurements, uninfluenced by reflections from the surrounding skin or the table on which the arm rested. The quantitative expression as formed by the four colors of the circumference of the top has the advantage that it may be

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[6] *Ibid.*

[7] Todd, T. W., Blackwood, Beatrice, and Beecher, Harry. Skin Pigmentation. The Color Top Method of Recording. *Am. J. Phys. Anthrop.*, vol. xi (1928), p. 188–91.

[8] Davenport, C. B. The Skin Colors of the Races of Mankind. *Natural History*, vol. xxvi (1926), p. 45–50.

[9] Todd, T. W., and Van Gorder, Leona. The Quantitative Determination of Black Pigmentation in the Skin of the American Negro. *Am. J. Phys. Anthrop.*, vol. iv (1921), p. 244.

[10] Herskovits, M. J. Age Changes in Pigmentation of American Negroes. *Am. J. Phys. Anthrop.*, vol. ix (1926), p. 321–322.

[11] Todd, T. W., and Van Gorder, Leona. *Op. cit.*, p. 240.

duplicated at will as a check on the validity of the original impression of the match between the blend of the whirling top and the skin, and also that it gives expressions which are directly comparable for any one series of individuals measured.

Todd, using Davenport's work as a base, made a study of the validity of the use of the color top as a method for the quantitative determination of the percentage of N pigmentation[12] in the skin of the American Negro.[13] He cites many difficulties to be overcome before accuracy and precision are attained. Fatigue of the color sense results from taking too many readings in succession, or pondering over the match in any one case. In addition, the blend produced by any given combination of sectors differs according to the speed at which the top is spun, darkening as the top slows down. Errors from both these sources are lessened if a prompt estimate is made of the match by glancing at the spinning top after whirling and judging by the instantaneous impression. Any one measurer automatically spins the top at a speed which does not vary appreciably from time to time, and hence this factor will be constant for one individual, though it may introduce constant differences in the results of two individuals. Todd and Van Gorder conclude as a result of their study that the proper precautions should reduce the observational error for the percentage of N pigmentation to between 2 and 5 per cent. Herskovits checked his own accuracy by measuring 48 boys; then remeasuring them on different days, under differing environmental conditions, and using old, intermediate, the new discs in various of the measurements. He found his observational error to be rather small, considering all the probable sources of error:[14]

N (black)—3.21 per cent.
R (red)—2.15 per cent.
Y (yellow)—2.19 per cent.
W (white)—1.44 per cent.

Thus Todd and Herskovits, working on Davenport's original method, have demonstrated the empirical validity of the color top as a practical method for securing a quantitative expression of the skin color of the American Negro. Methodologically, it is unsound. Todd in his latest paper concludes that "under the most careful correction it can

[12] In the remainder of this paper, N will stand for the black color.
[13] Todd, T. W., and Van Gorder, Leona. *p. cit.*, Op. 239–260.
[14] Herskovits, M. J. *Op. cit.*, p. 321–327.

be employed to give a relative record of which the accuracy is merely approximate. It is useful only pending the development of a more exact method".[15]

What precisely do those varying percentages of black, ox-blood red, yellow, and white which are the quantitative expression of the pigmentation of any individual mean? Do they describe something actually present in the skin of the Negro? Or are they merely conceptual descriptions of certain proportions of various colors, which, blended during the whirling motion of the top, produce a color similar to that of the skin of the individual being described? Davenport took the former position, asserting that the black constituent measures the melanin pigment; the "yellow probably is due to a lipochrome pigment so widespread in animals and found in the human hair and iris; the red is chiefly that of haemoglobin, and the white is reflected from the opaque skin".[16] Todd took the latter position, treating the various percentages as merely quantitative expressions of proportions of colors which, when blended through whirling, match the skin of the particular individual, disregarding any implications as to the correspondence of the various colors to specific component parts of the skin, except that the black (N) gives an indication of the amount of melanin pigment. Hence in utilizing his measurements Todd subtracts the 59 per cent of the percentage of ox-blood which is black from the percentage of red and adds it to the black sector reading. This correction makes all the readings in percentages of pure spectrum colors, so that all future work may be directly compared without further manipulation. Todd finds that the correction reduces the range of N to less than one-half the range in the uncorrected estimates, and makes possible the comparison and correlation of skin color with certain measurements of the skeleton. Before correction, correlation of skin color and these same skeletal features gave only chaotic and incomprehensible results.[17] For these reasons, the procedure of Todd and Herskovits has been followed in this study, and all percentages of black (N) pigmentation as given have been corrected by the addition of 59 per cent of the ox-blood red percentage of the same individual.

[15] Todd, T. W., Blackwood, Beatrice, and Beecher, Harry. *Op. cit.*, p. 203.

[16] Davenport, G. C. and C. B. Heredity of Skin Pigmentation in Man. *American Naturalist*, vol. xliv, p. 664.

[17] Todd, T. W. and Van Gorder, Leona. *Op. cit.*, p. 247. In this paper the authors do not state with what particular features of the skeleton skin pigmentation was correlated.

HISTORICAL REVIEW

The only quantitative study of the heredity of skin color previous to the present one is that of Charles B. Davenport on *The Inheritance of Skin Color in Negro-White Crosses.*[18] Davenport's plan was to seek for the key crosses from a Mendelian standpoint, secure as many generations as possible, and then analyze the heredity as it operates in specific families. On the basis of series measured in Jamaica, Bermuda and Louisiana, and using the black percentage uncorrected for the percentages of black in the ox-blood red, Davenport concluded that "there are two (double) factors, (A and B), for black pigmentation in the full-blooded Negro of the West Coast of Africa, and these are separately inheritable".[19] Davenport's study, while very valuable as a preliminary attempt to study pigmentation inheritance by quantitative methods, is not acceptable as a final analysis, if indeed any study based on color-top readings could possibly be. Todd has shown that when his readings were corrected for the percentage of black in the red, the bimodality of the curve for unmixed Negroes on which Davenport had based much of his genetic reasoning disappeared and a monomodal curve replaced it.[20]

Davenport's analysis is based on too few cases to furnish more than a fertile hypothesis, and, even were the number of cases greater, his methodology is not precise enough to prove many of his hypotheses. As an example, on page 24 he says that "there is a sex difference in the rate of development of pigmentation," the statement being based almost entirely on the percentage of N of the males and females in the one and two year groups. But when his data are corrected for the percentage of black in the red, the males have a higher percentage of N in the one year group and the females in the two, instead of the females having a higher percentage of N in both age groups as in his uncorrected figures.

	Uncorrected Percentages.		*Corrected Percentages.*	
Age	*Male*	*Female*	*Male*	*Female*
1 or less...	8.1 per cent N.	13.3 per cent N.	44.04 per cent N.	40.38 per cent N.
1 to 2.....	19.5 per cent N.	24.8 per cent N.	37.87 per cent N.	42.50 per cent N.

Again, "the increased variability of the F_2 as compared with the F_1 generation indicates the possibility of segregation". This state-

[18] Carnegie Institution of Washington, Publication No. 188.

[19] Davenport, C. B. *Op. cit.*, p. 13.

[20] Todd, T. W., and Van Gorder, Leona. *Op. cit.*, p. 258. For the original argument, see Davenport, C. B. *Op. cit.*, p. 10.

ment, so far-reaching in its implications as to the presence or absence of Mendelian inheritance in any population, is based on the total range in four supposedly mulatto-white crosses. Finally, concentration of frequencies at certain percentages of N, and therefore the conclusions based on it, is dependent on the validity of the failure to correct for the black percentage in the red. Chance has too much opportunity to operate in a small number of cases to attach great significance to slight differences.

Though Davenport's analysis of the inheritance of pigmentation cannot be accepted as final, it has been extremely valuable in demonstrating the practicability of the use of the color top to secure quantitative data for a study of this type. The methodological defects underlie both his work and ours, but a different analytical technique and a much larger series of measurements may make the reinvestigation of the problem of some value.

MATERIAL

The most comprehensive study yet made of the physical anthropology of the American Negro is that of Dr. M. J. Herskovits.[21] Series representing 5,659 individuals, of whom 3,378 were males and 2,281 females, and including individuals of all ages, were measured for from 18 to 30 traits. The measurements were taken from 1924 to 1927 in New York City; Washington, D. C.; Nashville, Tennessee; Tuskegee, Alabama; among the rural population of West Virginia; and, in 1915, a smaller series of 351 in eight cities of the South. Pigmentation readings were taken by the use of the color top for most of these individuals, but were available for a study of the inheritance of pigmentation only in those series in which measurements were taken for family groups. In addition to permitting the use of this mine of material, Dr. Herskovits has given frequent and invaluable assistance in the analysis of the problem and the techniques to be utilized.

The frequency distribution of the four series of measurements which were utilized in this study is given in Table I. Series I and II were measured by Dr. Herskovits and Mr. Louis King in the Harlem population of New York City in the winter of 1925–26. School children were chosen as representative of the general Harlem population, and measurements of both them and their parents taken to furnish

[21] For a partial list of the published results, consult the bibliography at the end of this study.

family groups. Series III was measured by Mr. Louis King in the summer of 1926 in the rural districts of West Virginia, and it, like the two Harlem series, consists of individuals of both sexes and all ages, in family groups. Series IV was measured by another assistant, Miss Zora Hurston, in the summer of 1926 among the well-to-do and professional Negroes of Harlem.[22]

TABLE I

*Number of American Negroes by Age Groups for the Series Used in This Study, Both Sexes**

AGE	SERIES I. HERSKOVITS' HARLEM		SERIES II. KING'S HARLEM		SERIES III. KING'S WEST VA.		SERIES IV. HURSTON'S HARLEM	
	♂	♀	♂	♀	♂	♀	♂	♀
1	4	1	..	..	3	1	4	2
2	9	7	3	1	5	8	6	4
3	5	10	8	9	13	10	4	2
4	15	8	9	3	19	9	8	3
5	31	32	7	7	7	15	3	6
6	113	43	16	9	7	18	4	5
7	152	63	11	10	7	19	3	3
8	150	50	12	13	9	14	3	...
9	148	17	7	9	10	23	...	2
10	144	17	14	11	8	16	1	2
11	157	15	33	7	13	23	1	4
12	157	16	61	12	13	26	1	2
13	186	4	94	7	18	11	...	6
14	144	5	88	7	15	17	2	1
15	99	6	86	7	3	12	2	3
16	36	7	18	7	3	6	5	6
17	17	5	8	7	6	7	4	7
18	6	4	2	4	2	6	3	3
19	...	1	5	3	3	3	8	2
20+	125	197	105	131	97	133	106	131

* This table includes all individuals for whom measurements were taken by Herskovits and his assistants, regardless of whether the pigmentation reading was used in this study.

[22] Herskovits, M. J. The Physical Form and Growth of the American Negro. *Anthropologischer Anzeiger*, Jahrg. iv, Heft 4, 1927, p. 293.

Genealogical data were secured for all the individuals and families of these series.[23] On the basis of the statements of the Negroes themselves as to the Negro-White composition of their ancestry, the entire population was divided by Herskovits into classes according to the amount of intermixture represented:

N—unmixed Negro.
NNW—more Negro than White.
NW—approximately equal amounts of Negro and White.
NWW—more White than Negro.

Despite the popular opinion that the Negro does not know his ancestry, Dr. Herskovits has shown by a consideration of physical traits that these genealogies are statistically valid in the sense that in the majority of those traits in which the two races differ, the mean and standard deviation of the NNW are more like the Negro; the NW intermediate; and the NWW more white.[24] Thus the material for this study contains not only quantitative expressions of the pigmentation of over 600 families, but statistically valid information concerning the approximate amount of race mixture represented in each individual and in each family.

Pigmentation readings were not available for every one of the individuals of the four series. Poor light, sun-burned skin, or other difficulties often made a reading impossible, or, if taken, valueless for our purpose. Only 4003 individuals were actually utilized for this study; 2173 for Series I; 1495 for Series II and III combined; and 365 for Series IV.

Each of these series included many individuals, usually school children, for whom there were no measurements of the family, either parents or brothers and sisters. These single individuals were utilized in the computation of growth curves, as they are obviously from the same general population as the families in the same series, but of course could not be utilized in the actual study of pigmentation inheritance. Eliminating them, there are 637 families, containing 2391 individuals. Table II shows the number of families in each series, the composition of the families as regards number of children, and whether measurements of one or both parents were secured.

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[23] Herskovits, M. J. Does the Negro Know his Father? A Study in Negro Genealogies. *Opportunity*, vol. iv (October 1926), p. 306–310.

[24] Herskovits, M. J. Variability and Racial Mixture. *American Naturalist*, vol. lxi (1927), p. 76.

COMPARABILITY OF THE DATA

The first question to be faced in utilizing these family groups in a study of the inheritance of N pigmentation is the extent to which the readings of different individuals are directly comparable. The color top is essentially a subjective device, the reading depending on the individual's perception of the blend of a particular combination of sectors of black, red, yellow, and white as matching the skin of a given Negro. The readings of any one individual are quite accurate, with an average observational error of only 2 to 5 per cent. But are the readings of two individuals comparable? And, if not, is there a consistent difference between them which, when eliminated, permits the readings to be treated as if made by one individual? Herskovits in his previous studies of pigmentation has never combined series measured by different observers.[25] In the four series of this study containing family groups, there are measurements taken by Herskovits (Series I); King (Series II and III); and Miss Hurston (Series IV). The desirability of having large numbers of measurements makes it necessary to combine these series. The measurements of Herskovits and Miss Hurston have as their only check readings of both on the same nine individuals. The difference in observation of N on the color top was only 1.33 per cent, Herskovits being darker. The small number of cases makes this of doubtful validity as an accurate measurement of their differences.[26] However, since Series IV, being selected well-to-do Negroes, and non-representative of the total Negro population of the country, is eliminated from the computation of growth curves, and is treated separately in all later stages, it is accepted as being comparable to Series I without any correction for the personal factor in measurement.

How do the measurements of King and Herskovits compare? Herskovits has previously made some tests as to the comparability of the two series. In a test sample of 100 boys, the readings for N are:[27]

King—mean percentage N, 72.92 $\pm$ 12.84.

Herskovits—mean percentage N, 73.65 $\pm$ 9.28.

But in the series measured on the Harlem population by the two men, King's results were consistently darker. In adult male American-born

[25] Herskovits, M. J. Age Changes in Pigmentation of American Negroes. *Am. J. Phys. Anthrop.*, vol. ix (1926), p. 321–322.

[26] Herskovits, M. J. The Anthropometry of the American Negro. MS., p. 19 and 43.

[27] *Ibid.*, p. 43.

TABLE II

Number and Composition of Families Used in This Study

PARENTS	NUMBER CHILDREN IN FAMILY	NUMBER OF FAMILIES BY SERIES				
		All	I	II	III	IV
One Parent—Father Only	1	35	6	18	7	4
	2	8	1	1	4	2
	3	3	...	...	3	...
	4	3	2	...	1	...
	5	1	...	...	1	...
	Total	50	9	19	16	6
One Parent—Mother Only	1	146	48	64	16	18
	2	62	22	21	14	5
	3	32	14	10	6	2
	4	13	3	5	3	2
	5	4	...	2	1	1
	6	2	...	2	...	...
	7	2	...	...	2	...
	Total	261	87	104	42	28
Both Parents	1	95	24	22	17	32
	2	84	37	16	8	23
	3	60	23	8	19	10
	4	41	14	17	8	2
	5	21	4	6	9	2
	6	15	2	6	6	1
	7	9	2	1	6	...
	8	...	...	...	...	...
	9	...	...	...	...	...
	10	...	...	...	...	...
	11	1	...	1	...	...
	Total	326	106	77	73	70
Total, All Families	1	276	78	104	40	54
	2	154	60	38	26	30
	3	95	37	18	28	12
	4	57	19	22	12	4
	5	26	4	8	11	3
	6	17	2	8	6	1
	7	11	2	1	8	...
	8	...	...	...	...	...
	9	...	...	...	...	...
	10	...	...	...	...	...
	11	1	...	1	...	...
	Total	637	202	200	131	104

Harlem Negroes, in which for all other traits there were no significant differences, the two men compared as follows:[28]

	No. Cases.	*Mean Percent N.*, $\pm \sigma$
King	40	79.10 ± 14.70
Herskovits	65	66.20 ± 13.44

Herskovits accounts for the similarity of the mean percentage of N in the case of the 100 boys in the test sample on the assumption that King, having compared his findings with those of Herskovits on the same persons at an earlier date when receiving instructions for the measurements, possibly was unconsciously influenced to judge color darker in the test cases. The much greater variability of King, ± 12.84 as opposed to that of ± 9.28 for Herskovits, lends plausibility to this theory.

These analyses of Herskovits justify us in accepting the fact that King's measurements for the same population were on the average darker than his own. The problem before us is to secure a quantitative expression of this difference. Boas dealt with this same question of the personal equation in measurements in his study of head form.[29] He assumed that the personal equation of the observers was distributed according to the laws of chance, the difference between the average of any one observer (a) and the true average (A) consisting of the accidental error (x) plus the personal equation (ϵ).

$$A - a = x + \epsilon$$

From this, Boas found that the general average may be obtained by giving the average obtained by each observer the weight:

$$W = \frac{1}{\frac{\sigma^2}{N} + (\epsilon^2)}$$

In our study, dealing principally with the measurements of only two men, the assumption that the errors due to the personal equations of various observers are distributed according to the laws of chance around a true average is inapplicable. Accordingly, the better procedure is to correct the readings of one observer (A) to those of the other (a) on the assumption that the difference between the average of the

[28] *Ibid.*, p. 117.

[29] Boas, Franz. Changes in Bodily Form of Descendants of Immigrants. Report of Immigration Commission, vol. xxxviii, p. 86–87.

two observers consists of the accidental error (x) and the personal equation (ϵ).

$$A - a = x + \epsilon$$

And, in large samples the accidental errors cancel out, so that we may use as a working formula:

$$A - a = \epsilon$$

Taking the adult males measured by the two men as samples, the mean pigmentation of Herskovits' measurements is 66.65 ± 13.98; of King's, 73.50 ± 13.11. King's readings are on the average 6.93 per cent darker than those of Herskovits, and, on the assumption that the accidental errors in observation either cancel out or are similar for the two men, we shall accept this as representing the personal equation in measurement. Since the observational error is ± 3 per cent, a difference of 6.93 may be taken for all practical purposes as 7 per cent.

In order to eliminate the personal equation, which may be assumed to represent a consistent difference between the measurements of the two observers, all King's readings were reduced 7 per cent, making them comparable to those of Herskovits. This is admittedly an empirical procedure, to be justified only by the consistency of the results for the two homogeneous series. Methodologically it is somewhat questionable on several counts. First, one must admit analogical reasoning if it is assumed that because the series measured by the two men show no significant differences in other traits, therefore they must be similar in relative darkness of skin. But the presumption tion is in favor of it, and this together with the very much greater variability of King when measuring the sample of 100 boys, and his greater darkness in the known Harlem series, justifies risking the initial correction provided it is checked at all later stages in the investigation by manipulating the various series separately. Second, the difference of 7 per cent is an average, and like all averages it is rather a conceptual symbol than characteristic of all the data entering into its computation. It represents an average of the consistent differences between the two series, while the difference in any individual case between the two readings might differ with the relative lightness or darkness of the individual being measured. For example, the percentage difference between the readings of a very dark individual, say between 75 and 80 per cent N, might possibly be greater than the difference between the readings for a very light individual, say between 25 and 30 per

cent N. However, with an original observational error of 3 per cent, the slight inaccuracy which might result from this hypothetical difficulty would be negligible.

After the correction the differences in the average pigmentation for any age group for the two men are fluctuating, and, where the number of cases is very large, slight. The approximation to equality is greater as the number of cases increases. For example, in the one year class, males, Herskovits' series is for four individuals, mean 80; King's, for two, 57.5. But when the 6 to 15 year groups for males are taken, where the number of cases is large, the series measured by the two observers are very similar, never differing more than ± 3 per cent.

TABLE III

Comparison of Age Changes for Males from 6 to 15 years of Age Measured by Herskovits and King respectively

AGE	HERSKOVITS' SERIES		KING'S SERIES		DIFFERENCE
	No.	Per cent N $\pm \sigma$	No.	Per cent N $\pm \sigma$	King-Herskovits
6	105	68.9 ± 13.50	21	67.8 ± 15.09	−1.1
7	150	70.9 ± 11.40	18	66.5 ± 15.24	−4.4
8	146	69.9 ± 13.17	19	68.8 ± 11.34	−1.1
9	148	71.1 ± 12.26	17	71.7 ± 12.96	+ .6
10	141	70.8 ± 11.43	20	65.9 ± 13.21	−4.9
11	156	72.6 ± 10.38	45	67.8 ± 12.14	−4.8
12	156	71.3 ± 11.85	76	70.6 ± 12.39	− .7
13	186	74.6 ± 8.94	108	72.4 ± 10.86	−2.2
14	144	72.6 ± 10.62	100	74.7 ± 9.93	−2.1
15	98	74.2 ± 12.15	90	75.0 ± 9.18	+ .8

On the basis of these results, we may conclude that the correction was valid, and that the difference in the readings on adults as a mass was a consistent difference approximately the same for all ages from 1 to 20, and for adults separated into five year groups.

AGE CHANGES IN PIGMENTATION

In an analysis of the inheritability of a variable trait, the reduction of all readings to those of one specific age group is a *sine qua non* of

scientific procedure. That skin color does change with age has been asserted by most students. Davenport found that "the proportion of black in the skin increases to about the age of 10 or 12, and then slowly diminishes."[30] Todd and Van Gorder state that "the period of darkest skin is 10–19 years, but the average of this period is fairly steadily maintained until the late forties, after which there does seem to be a diminution in the blackness of the skin."[31] Herskovits made a study of age changes in pigmentation in the series measured by himself alone, and found that there was a fairly steady increase in the percentage of N (with the exception of 11½ years) from the 5½ year average of 64.23 ± 14.7 to the 17½ year average of 74.07, while the average of the adults was 66.40 ± 13.85.[32]

Obviously the results of none of these studies are quantitatively precise enough to permit the correction of the readings of all age groups to those of one specific age. Even if they were, the percentage change with age might be different for Negro populations of different origins and containing different amounts of racial intermixture. For these reasons, growth curves were made for Series I, II, and III, both separately and combined. Series IV, consisting of the measurements of a selected well-to-do and professional Harlem group, was excluded as it is admittedly not a random sample of the population and would overweight those age groups in which it contained many individuals with a larger proportion of lighter individuals than are typical of the normal age distribution in the population as a whole.

Since the entire population, and hence any given age group within the population, is chosen at random, the distribution of different percentages around the mean for that age group should be approximately the same in each age group, given a sufficient number of cases.[33] On this assumption, the truth of which is substantiated by the standard deviations for the various age groups, changes in the mean pigmentation from year to year could be taken as indicating age changes. Means were secured for each age and each sex from 1 to 20; by five year periods from 20 to 60; and for all individuals over 60, for Series I, II and III separately. Taking the similarity of mean pigmentation and the general consistency of change as indicating fairly homogeneous samples,

[30] Davenport, C. B. *Op cit.*, p. 7.

[31] Todd, T. W., and Van Gorder, Leona. *op. cit.*, p. 259.

[32] Herskovits, M. J. Age Changes in Pigmentation of American Negroes. *Am. J. Phys. Anthro.*, vol. ix (1926), p. 325–326.

[33] *Ibid.*, p. 321–327.

the two series of King and the one of Herskovits were combined and the mean and standard deviation for the total number of males and females computed separately. The data thus combined confirm the conclusions of Davenport[34], Todd,[35] and Herskovits,[36] that there are no sex differences in the pigmentation of the American Negro. Wherever the number of cases in any one age is large, the mean of males and females tends to approach. Such differences as exist are fluctuating, sometimes one and sometimes the other sex being darker.

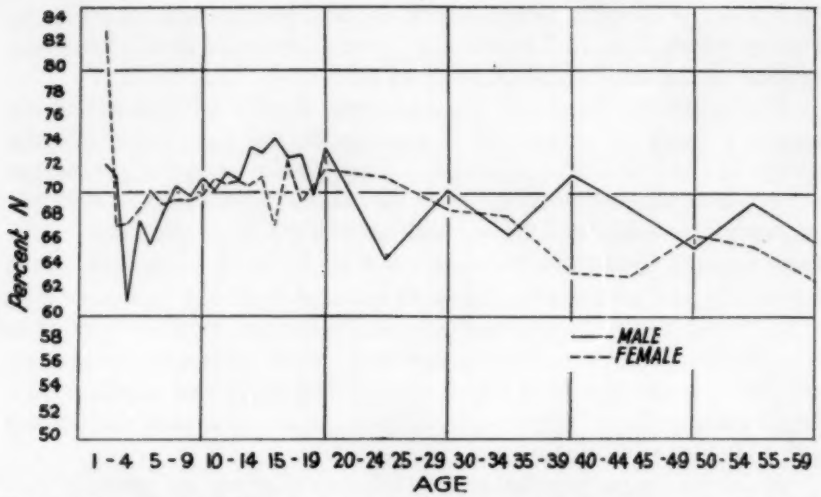

FIG. 1. COMPARATIVE PERCENTAGE OF N. PIGMENTATION OF MALES AND FEMALES BY AGE GROUPS, KING'S AND HERSKOVITS' COMBINED SERIES

The curves for males, females, and the combined sexes were smoothed by the use of a three year running average of the means, extended by a projection of the trends to the upper and lower years. The smoothed means of each age for males, females, and the combined sexes can be directly compared in Table V. Since the smoothed curves were so nearly regular, and so nearly alike for the two sexes, it seemed probable that the minor irregularities in the trend were due to chance

[34] Davenport, C. B. *Op. cit.*, p. 47.

[35] Todd, T. W., and Van Gorder, Leona. *Op. cit.*, p. 259. Also Todd, T. W., Blackwood, Beatrice, and Beecher, Harry. *Op. cit.*, p. 195–196.

[36] Herskovits, M. J. Social Selection in a Mixed Population. *Proceedings of the American Academy of Sciences*, Vol. XII (1926), p. 589.

TABLE IV

Age Changes in Percentage of N Pigmentation of the American Negro, Both Sexes and All Series Measured by Herskovits and King

AGE GROUP	MALES		FEMALES		ALL	
	No.	Mean ± σ	No.	Mean ± σ	No.	Weighted* Mean
1	6	72.50 ±	1	83.00 ±	7	70.70
2	16	71.40 ± 8.19	15	67.01 ± 12.45	31	66.75
3	26	61.24 ± 12.24	29	67.28 ± 13.20	55	66.80
4	43	67.09 ± 14.67	18	68.84 ± 9.60	61	67.00
5	44	65.90 ± 14.75	54	70.03 ± 11.94	98	68.50
6	126	68.72 ± 13.77	68	69.23 ± 9.75	194	69.40
7	168	70.49 ± 11.67	92	69.77 ± 11.34	260	69.70
8	165	69.77 ± 12.69	76	69.53 ± 10.29	241	70.30
9	165	71.15 ± 12.69	47	69.86 ± 10.50	212	70.30
10	161	70.16 ± 11.79	43	71.15 ± 11.34	204	70.90
11	201	71.54 ± 10.98	43	70.73 ± 10.20	244	71.00
12	232	71.09 ± 12.08	51	70.94 ± 10.55	283	72.10
13	294	73.81 ± 10.08	22	70.46 ± 9.69	316	72.70
14	244	73.45 ± 10.47	28	71.33 ± 11.26	272	73.50
15	188	74.57 ± 10.83	23	66.83 ± 11.69	211	73.40
16	58	73.02 ± 10.47	19	72.59 ± 7.05	77	73.30
17	34	73.13 ± 10.47	19	69.63 ± 11.07	53	71.40
18	6	69.80 ±	13	70.31 ±	19	71.00
19	7	73.85 ±	6	72.00 ±	13	71.80
20–24	9	64.67 ±	35	71.43 ± 7.36	44	69.70
25–29	17	70.31 ± 11.89	53	68.87 ± 12.16	70	68.50
30–34	39	66.56 ± 12.77	123	68.10 ± 12.41	162	66.00
35–39	67	71.49 ± 12.53	120	63.74 ± 14.13	187	66.80
40–44	76	68.99 ± 13.78	76	63.26 ± 13.89	152	66.30
45–49	49	65.71 ± 13.06	48	66.68 ± 12.29	97	66.40
50–54	23	69.44 ± 12.75	26	65.57 ± 12.86	49	66.40
55–59	13	66.17 ±	7	62.85 ±	20	66.02
60	14	63.50 ±	21	61.43 ± 10.54	35	62.00

* Based on three-year moving average.

and the small numbers involved. Considering the initial error of observation and the relatively rough measurements in general, it was decided that it would be spurious accuracy to fit a curve mathematically to the curve of pigmentation, changing its slope as it does with age. Hence a curve was drawn free-hand to fit the smoothed curve for the

TABLE V

Age Changes in Percentage of N Pigmentation of the American Negro, Actual Data Smoothed and Idealized

AGE GROUP	SMOOTHED			IDEAL (FREE-HAND)
	Male	Female	Combined	
1	71.87	68.89	70.74	66.00
2	66.01	67.55	66.75	66.50
3	66.10	67.60	66.76	67.00
4	65.27	69.03	67.04	68.00
5	67.79	69.46	68.46	68.50
6	69.20	69.66	69.39	69.00
7	69.75	69.50	69.67	69.50
8	70.50	69.70	70.26	70.00
9	70.40	70.05	70.31	70.25
10	71.01	70.57	70.92	70.50
11	70.99	70.93	70.98	71.00
12	72.30	70.75	72.08	71.25
13	72.90	70.92	72.66	71.50
14	73.90	69.64	73.51	72.00
15	73.86	70.17	73.40	72.50
16	74.08	69.47	73.26	72.25
17	72.90	70.89	71.42	72.00
18	72.80	70.20	71.02	71.75
19	72.68	71.12	71.79	71.25
20–24	67.41	70.32	69.67	69.50
25–29	67.27	68.84	68.47	68.00
30–34	65.01	66.45	66.01	67.00
35–39	69.39	65.30	66.78	66.50
40–44	69.03	64.16	66.30	66.25
45–49	67.97	64.78	66.36	66.00
50–54	66.78	66.01	66.41	65.75
55–59	66.92	64.70	65.92	65.50
60+	64.34	61.16	62.43	64.00

entire population. This ideal curve, the values for which are given in Table V, was tested by fitting it to the smoothed curve for the sexes taken separately. Deviations above and below it are random in the curves secured by plotting it above the actual means of the combined sexes or male and female separately. It may be taken as representing the age changes in pigmentation for the American Negro population of the amount of intermixture and the average pigmentation of the various series Herskovits used in his studies.

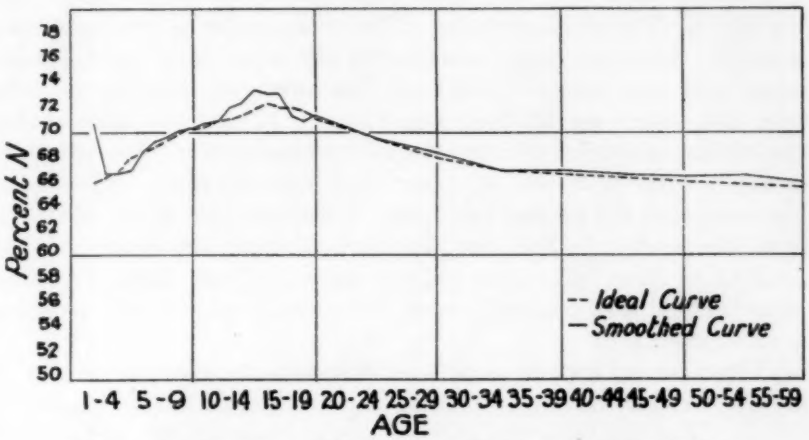

Fig. 2. Age Changes in Pigmentation of the American Negro

In summary, the percentage of N pigmentation of the Amercian Negro increases quite regularly until adolescence, reaches a peak at the age of 14, 15, and 16; decreases quite rapidly until the age of 30 to 35; and then decreases slowly the remainder of life. The precise figures for various ages are of use for our particular study, but of little value as a general statement of age changes, as the average for any given age obviously depends upon the percentage of intermixture and the general lightness or darkness of the population one is considering. However, if these 3652 individuals may be taken as representative of the fairly homogeneous physical type of the American Negro, then for the American Negro there is an increase from the one year mean of 66 per cent to the 15 year mean of 72.50 per cent; a decrease to the 30–34 mean of 67 per cent; and then a slow decrease to the mean of 64 per cent for all individuals over 60.

The ideal curve as thus secured was used as the basis for correcting readings of all individuals to those of the age group 35–39, the adult group with the largest number of individuals in it. There is a slight source of error in this method of correction in that it, just as the correction of the readings of different observers, represents an average of the pigmentation changes from year to year which might and possibly would not represent the precise amount of change for any particular individual in the population. An individual with 32 per cent N at the age of 17 might not lighten as much with increasing age as one with 76 per cent N. Two individuals each with 76 per cent N at the age of 17 might lighten by different amounts by the time they were 35. However, again considering the 3 per cent observational error, and the lack of statistical bias involved, any inaccuracies from this source are of slight significance. It might be desired that the technique of color determination were capable of yielding measurements precise to 1/10th of 1 per cent, and absolutely objective in the sense that the personal equation of the measurer would not influence the result. In this case, the correction for age changes would need to be done by a more refined technique, but, given the large observational and personal error, the present method of correction is as valid as any.

After correction for personal differences in the observers and for age changes, there is a range of only 3.37 per cent in the mean percentage N of the three non-selected series, while the selected series is 10.46 per cent below the lowest of the other three.

Series	*No. Individuals*	*Mean*	*Standard Deviation*
I	739	68.45	±11.61
II	714	65.05	±13.33
III	573	64.88	±12.75
IV	365	54.29	±10.91
Total:	2391	64.37	±13.02

Taking the frequency distribution in genealogical classes, it is interesting to note the massing in the upper ranges of pigmentation in the N group, and the progression until the massing in the NWW group is in the smaller percentages of N (Table VI). The decreasing mean pigmentation and increasing variability as the amount of white intermixture increases is in accordance with theoretical expectations if both the genealogies and the readings with their corrections for various factors were valid. This fact, together with Herskovits' demonstra-

tion through a study of physical traits that the genealogies secured for his group of 539 Howard University Negroes were statistically valid,[37] permits us in our later analysis of heredity to utilize these genealogical classes as indicating roughly the percentage of intermixture in various groups.

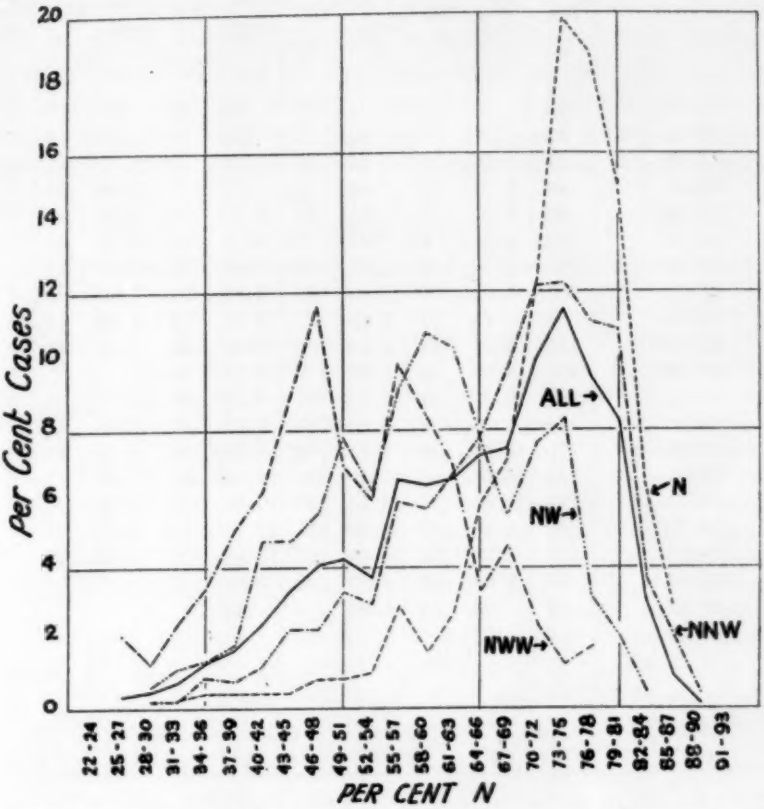

FIG. 3(a). PERCENTAGE FREQUENCY DISTRIBUTION OF AMERICAN NEGROES BY PER CENT OF N PIGMENTATION, BY GENEALOGICAL CLASSES

Attention may be called here to the greater variability of the NWW as contrasted to the NW (Table VI). Discussion of this point will be reserved until later when the question of the Mendelian inheritance of pigmentation is being considered.

[37] Herskovits, M. J. The American Negro: A Study in Race Crossing. *New York*, Alfred A. Knopf (1928), p. 18–33.

TABLE VI

Percentage of N Pigmentation by Genealogical Classes for All Series Used in This Study, After Correction for Personal Equation and for Age Changes

PERCENTAGE OF N	ALL CLASSES		N		NNW		NW		NWW	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
25–27	8	.33	..	.....	...	.....	..	.....	8	2.05
28–30	10	.42	..	.....	2	.19	3	.67	5	1.28
31–33	17	.71	1	.20	2	.19	5	1.12	9	2.30
34–36	31	1.30	2	.41	9	.85	7	1.57	13	3.32
37–39	40	1.67	2	.41	8	.75	8	1.80	22	5.63
40–42	60	2.51	2	.41	13	1.22	21	4.72	24	6.14
43–45	81	3.39	2	.41	23	2.16	21	4.72	35	8.95
46–48	97	4.06	4	.81	23	2.16	25	5.62	45	11.51
49–51	101	4.22	4	.81	35	3.29	35	7.86	27	6.91
52–54	89	3.72	5	1.02	31	2.92	28	6.29	25	6.39
55–57	157	6.57	14	2.84	63	5.93	41	9.21	39	9.97
58–60	151	6.32	8	1.63	62	5.83	48	10.78	33	8.44
61–63	157	6.57	13	2.64	71	6.68	46	10.33	27	6.91
64–66	174	7.28	29	5.89	83	7.81	35	7.86	27	6.91
67–69	178	7.44	35	7.11	104	9.78	26	5.84	13	3.32
70–72	241	10.07	60	12.19	129	12.13	34	7.64	18	4.60
73–75	274	11.45	98	19.91	130	12.22	37	8.31	9	2.30
76–78	230	9.62	93	18.90	118	11.10	14	3.15	5	1.28
79–81	197	8.24	74	15.04	107	10.06	9	2.02	7	1.79
82–84	74	3.09	32	6.50	40	3.76	2	.45	.....	.....
85–87	22	.92	14	2.84	8	.75	.....	.....	.....	.....
88–90	2	.08	..	.....	2	.19	.....	.....	.....	.....
			N		*NNW*		*NW*		*NWW*	
Total:	2391		492		1063		445		391	
Mean:	64.37		72.74		67.19		58.73		52.73	
$\pm\sigma$	$\pm$13.02		$\pm$8.93		$\pm$11.21		$\pm$11.12		$\pm$12.35	

ANALYSIS OF THE METHOD OF INHERITANCE

Having the data relatively comparable both as to the personal factor in measurement and the age changes during the life of the individual, we may proceed with the analysis of the method of inheritance itself. The first hypothesis made as to the nature of the inheritance of pigmentation was that it was a blend. If there is a true blend, and not merely Mendelian heterozygotes, then diverse parents produce offspring intermediate between them in type, with no segregation of

the parental traits in the second filial generation. If this conception of the fusion of parental hereditary qualities holds in the concrete instance, the average pigmentation of the offspring must be the same as the average pigmentation of the parents. The pigmentation of the sons, daughters, and all children for each family was averaged separately, and the pigmentation of the parents averaged in those cases in which the measurements of both parents were available. Tables VII(a), VIII, and IX give the average value of offspring, sons, and daughters for the average percentage of the parents, for the fathers, and finally by mothers, by 3 per cent groups, all series combined. Table VII (b) gives the average per cent N of the lighter

TABLE VII(a)

Average Percentage of N Pigmentation of Children of Parents of Varying Percentages of N, All Series

AVERAGE OF PARENTS	AVERAGE OF ALL OFFSPRING		AVERAGE OF SONS		AVERAGE OF DAUGHTERS	
	No. Fam.	Per Cent N	No. Fam.	Per Cent N	No. Fam.	Per Cent N
34–36	2	42.37	2	48.08	2	36.60
37–39	3	43.76	2	45.15	1	41.00
40–42	6	45.69	5	46.18	5	46.40
43–45	12	43.01	9	43.42	6	43.40
46–48	12	49.42	9	51.69	5	43.40
49–51	18	50.24	14	50.96	14	49.61
52–54	21	53.78	18	53.62	14	54.08
55–57	22	58.31	19	58.23	14	57.55
58–60	28	60.19	24	61.61	23	58.22
61–63	18	58.51	15	59.87	13	57.47
64–66	37	64.08	33	63.25	22	65.76
67–69	29	67.36	21	68.62	20	66.50
70–72	31	69.74	26	69.88	19	70.38
73–75	20	72.05	17	73.27	12	70.80
76–78	32	74.42	30	74.27	27	73.89
79–81	24	77.55	23	77.92	15	77.41
82–84	8	78.17	8	78.47	4	76.81
85–87	3	80.87	3	80.16	1	81.50
88–90						
Regression on Parents (3 Per Cent Group).....	..	2.43	..	2.78	..	2.59

parent and of the offspring for darker parents of various percentages of N, and shows that in the lower ranges, the pigmentation of the offspring is either nearly equal to or greater than that of the darker parent; from say 53 per cent N to 77 per cent N, it is approximately

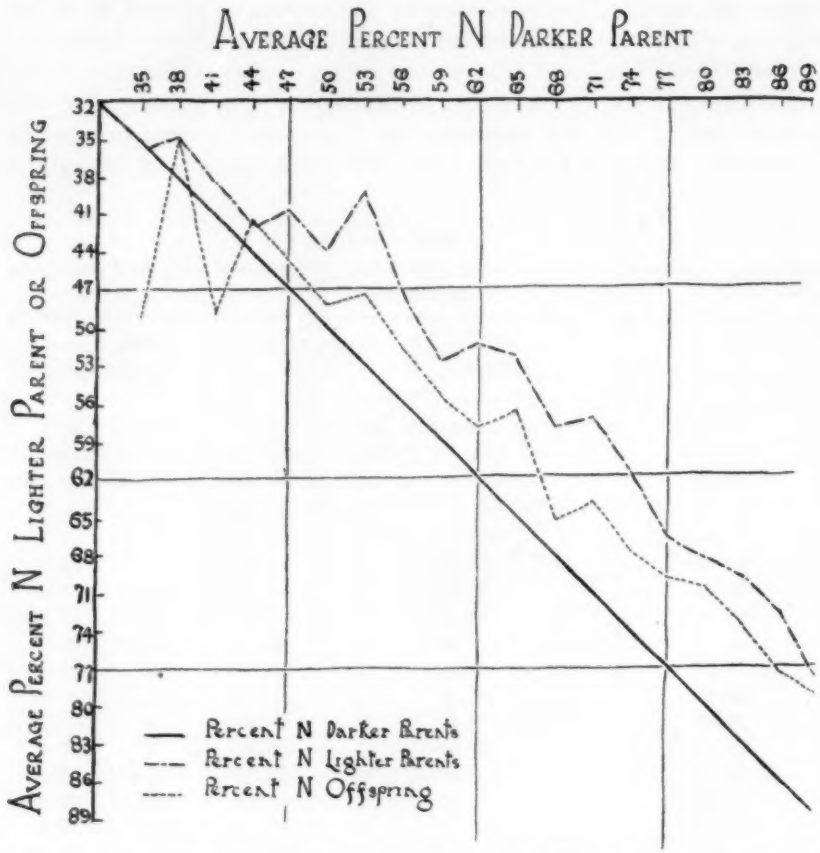
Average Percent N Darker Parent
35 38 41 44 47 50 53 56 59 62 65 68 71 74 77 80 83 86 89
Average Percent N Lighter Parent or Offspring
32 35 38 41 44 47 50 53 56 59 62 65 68 71 74 77 80 83 86 89
Percent N Darker Parents
Percent N Lighter Parents
Percent N Offspring

Fig. 3(b). Relation of Average Percent N Pigmentation of Offspring to that of their Fathers and Mothers

intermediate; and above 77 per cent N, it approaches more nearly to the average of the lighter parent. The data for this table were treated, first holding males as the independent variable, and then females; as there were no significant differences, it was permissible to hold the darker parent as the independent variable, regardless of

sex. The same characteristic phenomena occur also if the lighter parent is held instead of the darker.

TABLE VII (b)

Average Percentage N Pigmentation of Offspring of Fathers and Mothers of Varying Percentages of N

PERCENT N DARKER PARENT	NO. FAM.	AVERAGE PERCENT N	
		Lighter Parent	Offspring
34–36	1	36.00	49.62
37–39	2	34.62	34.96
40–42	2	38.37	48.77
43–45	6	42.00	41.91
46–48	7	40.71	44.50
49–51	13	43.35	48.03
52–54	3	39.25	47.50
55–57	22	47.04	51.42
58–60	18	52.23	55.26
61–63	17	51.28	57.56
64–66	20	50.51	56.46
67–69	23	57.76	65.16
70–72	30	57.32	63.66
73–75	41	61.54	67.96
76–78	36	66.70	70.32
79–81	42	68.31	70.68
82–84	32	69.79	73.79
85–87	10	72.57	77.46
88–90	1	78.00	79.00

These averages furnish a final means of checking on the possibility of sex-differences or sex-dominance in the inheritance of pigmentation. The means and standard deviations of the various age groups show a remarkable similarity in the average percentage of males and females in the various age groups, and there is no consistent difference in the averages of sons and daughters for the given percentage parent. The similarity of the regression coefficients for both sons and daughters in all cases offers still further proof of the absence of sex differences in pigmentation in the American Negro population.

What do these series of averages indicate as to the resemblance of parents and children? In Table VII the average of the offspring is above that of the parents in the lower percentages; approximately the same from 50 to 69 per cent; generally falling within the same

3 per cent group as the average percentage of the parents; and below that of the parents from 69 per cent to 87 per cent. This eliminates the theory of blending inheritance and suggests regression of the

TABLE VIII

Average Percentage of N Pigmentation of Children of Fathers of Varying Percentages of N, All Series

AVERAGE OF FATHERS	AVERAGE OF ALL OFFSPRING		AVERAGE OF SONS		AVERAGE OF DAUGHTERS	
	No. Fam.	Per Cent N	No. Fam.	Per Cent N	No. Fam.	Per Cent N
28–30	3	59.66	2	66.00	2	52.75
31–32	2	44.11	2	45.96	2	42.09
34–36	6	53.24	2	53.32	4	52.54
37–39	6	49.02	6	47.50	3	51.27
40–42	12	52.63	11	53.14	6	56.78
43–45	12	46.57	8	49.74	8	44.75
46–48	14	51.39	11	49.66	9	52.26
49–51	7	52.07	6	53.08	2	45.37
52–54	13	58.07	10	60.52	10	58.92
55–57	21	53.92	14	57.70	17	51.94
58–60	19	56.05	16	57.78	14	55.65
61–63	21	60.97	17	59.27	12	64.35
64–66	24	61.03	21	60.81	11	57.58
67–69	26	66.79	24	67.18	14	67.48
70–72	27	66.19	21	65.95	18	64.11
73–75	37	68.22	32	68.27	25	67.67
76–78	36	71.25	31	73.85	25	68.23
79–81	55	71.24	42	71.96	38	71.93
82–84	23	73.56	20	75.86	14	71.46
85–87	11	76.53	8	78.30	8	75.74
88–90	1	79.00	1	79.00	.....	.....
Regression on Fathers (3 per cent Groups).....	..	1.48	..	1.43	..	1.45

average percentage of the offspring to the mean pigmentation of the group. Regression coefficients of offspring, sons, and daughters on parents, fathers and mothers respectively were computed by the method of least squares.[38] They furnish a summary expression of the

[38] Secrist, Horace. An Introduction to Statistical Methods. *New York*, The Macmillan Co. (1925), p. 445.

relationship strikingly shown in Tables VII to IX, and illustrated in the following figure, the data for which are taken from Table VII. There is a decided tendency for the offspring of any family to differ

TABLE IX

Average Percentage of N Pigmentation of Children of Mothers of Varying Percentages of N, All Series

AVERAGE OF MOTHERS	AVERAGE OF ALL OFFSPRING		AVERAGE OF SONS		AVERAGE OF DAUGHTERS	
	No. Fam.	Per Cent N	No. Fam.	Per Cent N	No. Fam.	Per Cent N
25–27	1	21.33	1	20.10	1	27.50
28–30	4	53.70	4	53.70	.....	.....
31–33	6	48.91	4	53.72	4	50.69
34–36	6	49.78	6	50.83	1	43.30
37–39	9	51.94	7	53.60	3	42.45
40–42	21	50.40	14	55.82	16	49.10
43–45	22	51.17	15	52.51	15	50.91
46–48	29	54.16	24	57.11	20	52.34
49–51	42	52.39	31	53.87	23	49.63
52–54	22	55.32	17	56.51	16	56.93
55–57	41	61.20	33	60.74	27	61.15
58–60	31	61.76	27	61.39	21	64.03
61–63	38	63.86	25	65.56	23	60.16
64–66	38	66.06	32	66.46	23	65.88
67–69	38	65.79	29	66.40	20	65.65
70–72	45	68.35	39	68.64	28	68.40
73–75	58	69.06	45	69.36	39	69.54
76–78	48	70.69	41	72.10	27	75.02
79–81	48	74.11	41	75.31	30	72.72
82–84	32	76.18	30	76.59	21	75.92
85–87	6	75.42	4	76.25	4	74.60
88–90	1	80.50	1	80.50	.....	.....
Regression on Mothers (3 per cent Groups).....	..	1.84	..	1.77	..	2.81

less from the modal value of the population than does the average value of the parents, or the value of either father or mother taken individually. Table X summarizes the regression coefficients for the series of averages in Tables VII to IX.

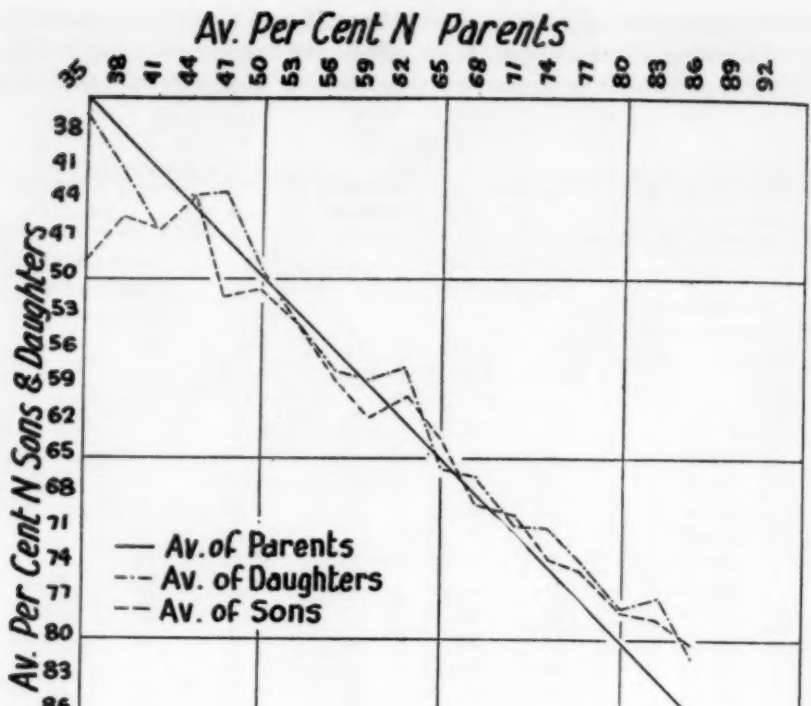

FIG. 4. REGRESSION OF SONS AND DAUGHTERS ON AVERAGE PERCENTAGE N PIGMENTATION OF PARENTS

TABLE X

Summary of Regression Coefficients for All Series

REGRESSION ON (3 PER CENT GROUPS)	REGRESSION COEFFICIENTS		
	All Offspring	Sons	Daughters
Av. of Parents	2.43	2.78	2.59
Fathers	1.48	1.43	1.45
Mothers	1.84	1.77	2.81

In the above table, the regression coefficient of all offspring on mothers is between that for sons and daughters taken individually;

on fathers, above; and on the average of the parents, below. Logically it would seem that the regression coefficient for all children should be intermediate between those for sons and daughters, since the averages used for securing the coefficient for all children are an average of all the individual family averages in any frequency group. But it must be remembered that there are many families in which the children are of one sex entirely. For example, where the percentage of N is 70–72, there are 31 families with offspring of both sexes and a mean pigmentation of 69.74; 26 with sons of a mean pigmentation of 69.88; and 19 with daughters of a mean pigmentation of 70.38. Since this condition, in which the average of all children for any average per cent N parents may be greater or less than that of the average of either sons or daughters taken separately for the same average per cent N parents, is secured in many of the various frequency groups which form the data for the computation of the regression coefficients, it follows that the cofficient of regression of all offspring may be either greater, less, or intermediate between that for sons and daughters taken separately.

The correspondence between the actual data and the regression line is very close. For the regression of all offspring on parents, the average difference of the actual data from the regression line is only 1.63 per cent; of sons on parents, 2.43 per cent; and of daughters on parents, 1.52 per cent.

The regression coefficients of offspring on the various percentages of fathers and mothers, taken separately, are greater than those on the average percentage of the two parents. Here again there are no consistent differences as between the regression of either sons or daughters on the same sex parent, or the regression of both on parents of different sex. There is no indication of sex linkage or sex dominance in inheritance.

The regression of offspring by genealogical classes shows the same tendency for the offspring to deviate less from the mean of the genealogical class than do the parents. In the unmixed Negro, the regression coefficient of offspring on parents is 3.31, but the validity of this regression is questionable because of the number of cases in the extreme percentages. In the NNW group, the regression coefficient is 2.38; in the NW, 2.36; and in the NWW, 1.76. The correspondence between the average percentage of parents and the average percentage of the children becomes less as the amount of racial intermixture increases. The greater the racial heterogeneity, the

less stable the family lines and the more they tend to regress to the modal pigmentation percentage of the group.

This analysis of the data secured by the use of averages offers the following evidence pertaining to the resemblance of parents and children:

1. There is no sex difference in the resemblance of offspring to parents in the series of averages.

2. The average percentage of N of the offspring tends to conform quite closely to the average of the parents in the middle ranges of pigmentation; to be above it in the lower percentages; and below it in the higher, as would be expected.

3. The changes in the pigmentation of parents and children tend to bear a systematic relationship to each other which may be expressed by means of the coefficient of regression, children almost always differing less from the mean pigmentation of the group than their parents, which is according to expectations.

4. Regression coefficients of both sons and daughters taken separately show no consistent differences, nor is there any consistent difference whether the regression be on father or mother.

5. The regression coefficient, and hence the regular simultaneity of change, is less as the amount of white intermixture increases: 3.31 in the N; 2.38 in the NNW; 2.36 in the NW; and 1.76 in the NWW.

Apparently these series of averages, while suggestive, do not give us definitive evidence as to the precise method of the inheritance of N pigmentation in the American Negro. Another method of attack is through the comparative variability of groups representing different amounts of racial intermixture. The general biological assumption has been that when race crossing occurs, the hybrid offspring is more variable than either uncrossed race.[39] Obviously if the Mendelian hypothesis as commonly conceived is to hold for the inheritance of any particular trait in which the two inter-breeding races differ widely, the variability of a hybrid offspring must be greater than either parent race.

What are the actual facts as regards variability in Negro-White crosses relative to the variability of the unmixed Negro? First, we may compare the total variability for the various genealogical classes, expressed in terms of the mean square deviation of the individuals of

[39] e.g., Castle, W. E. Biological and Social Consequences of Race Crossing. *Am. J. Phys. Anthrop.*, vol. ix (1926), p. 152.

a group from the mean of that group. Herskovits has shown the standard deviation of the genealogical classes of Howard University students to increase progressively as the intermixture with the white race becomes greater.[40]

Genealogical Class	*Mean Percentage N* $\pm \sigma$
N	75.5 ± 10.3
NNW	68.3 ± 11.6
NW	61.2 ± 12.0
NWW	48.7 ± 13.7

In the four series used for this study, after correction for the personal factor in measuring, and for age changes, the standard deviation is found to show the same progressive increase from the N to the NWW class, with a slight slump in the NW as compared to the NNW. This may perhaps be taken to be in accord with expectations

Genealogical Class	*No. Individuals*	*Mean Percentage N* $\pm \sigma$
N	492	72.74 ± 8.93
NNW	1063	67.19 ± 11.21
NW	445	58.73 ± 11.12
NWW	391	52.73 ± 12.35
Total:	2391	64.37 ± 13.02

if segregation in inheritance of pigmentation were occurring. The greater variability of the NWW class, here as well as in the fraternal variability, when compared to the NW class, which is theoretically more racially heterogeneous, is rather difficult of interpretation. Until further research has been carried out in the pigmentation of various Caucasoid groups, and their variability determined, any explanation we might offer would be too speculative to possess any value.

But the total variability of any population may be conceived as consisting of two types of variability, that within the families of the population, and that between the various family lines. Fraternal variability, or the differences of brothers and sisters within the family, indicates whether segregation, blending, or some other phenomenon as yet undescribed is occurring in the inheritance of any specific trait in which the parental stocks differ. The variability of family lines indicates the homogeneity or heterogeneity of a population for a particular trait. If a population is of unmixed racial stock, or if there

[40] Herskovits, M. J. The American Negro: A Study in Race Crossing. P. 15.

has been early intermixture followed by long-continued in-breeding, there will be slight variability between family lines, as all families may be regarded as having essentially the same ancestors.[41] Hence, provided intermixture has been relatively recent, the family variability of any given group as regards N pigmentation will show the racial heterogeneity of that group, and thus the amount of variability that would be expected within the families of that group were Mendelian segregation occurring. The relation of fraternal and family variability in this American Negro population, known to be racially heterogeneous, should indicate the types of matings which have been taking place in the majority of cases, and this in turn should aid us in deciding what types of variability to expect in various groups in accordance with the various hypotheses as to the method of inheritance which we may later examine.

The method of determining the variability of family lines has been worked out by Professor Franz Boas[42], who started with the assumption that the deviation of any individual from the general mean of the population of which he is a part (x) will consist of his deviation from the average of his family (ξ), and the deviation of that average itself from the general mean (η).

$$(1)\quad x = \xi + \eta$$

The mean square variability will then equal:

$$(2)\quad [x^2] = [\xi^2] + [\eta^2]$$

the brackets indicating averages. Developing this formula we find that S^2_f, the variability within the families, is equal to:

$$(3)\quad \sigma^2(1 - r)^{43}$$

However, starting from a slightly different point, Professor Boas has developed a somewhat simpler approach to the problem which

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[41] Herskovits, M. J. On the Negro-White Population of New York City; The Use of the Variability of Family Strains as an Index of Heterogeneity or Homogeneity. *Proceedings, XXIe Congres International des Americanistes*, Hague, 1926, p. 5–12.

[42] Boas, Franz. On the Variety of Lines of Descent Represented in a Population. *Amer. Anthro.* (N.S.), vol. xviii (1916), p. 1–9.

[43] Herskovits, M. J. *op. cit.*, p. 6–7; also see the paper by the same author: A Further Discussion of the Variability of Family Strains in the Negro-White Population of New York City. *Jour. Amer. Statistical Ass'n* (N.S.) vol. xx (1923), p. 380–389, for a detailed development of this formula.

eliminates the necessity of computing the coefficient of fraternal correlation, r,[44] thus lessening the actual amount of labor involved while securing the same results. If we assume the deviation of an individual (i_1) to be composed of his deviation from the average of his family (x_1), and that of this average from that of the entire population, (η), then:

$$(1)\ i_1 = \eta + x_1$$

The deviation of his brother will be:

$$(2)\ i_2 = \eta + x_2$$

and (i_3), (i_4) etc. for each member of the fraternity. Subtracting,

$$(3)\ (i_1 - i_2) = (\eta + x_1) - (\eta + x_2).$$
$$= x_1 - x_2.$$

The mean square variability of this will of course represent the variation within the family, or

$$(4)\ [(i_1 - i_2)^2] = [(x_1 - x_2)^2]$$
$$= [x_1^2] - 2[xx] + [x^2].$$

However,

$$(5)\ [x^2] = \sigma^2 \text{ of the entire population.}$$

And:

$$(6)\ [xx] = r\sigma^2$$

Hence:

$$(7)\ [(i_1 - i_2)^2] = 2\sigma^2 - 2r\sigma^2$$
$$= 2\sigma^2\,(1 - r).$$

which is, of course, the same.

The error of the fraternal variability is derived from the mean square variability of the difference,

$$(8)\ [x_1 - x_2 - x_3 \ldots x_n]^2$$

assuming families of an infinite number of children, and is found to be:

$$(9)\ \frac{\sigma^2(1 - r)}{n\,(n - 1)}$$

The weighting to be given families of differing numbers of children, therefore, will be $n(n - 1)$, n representing the number of children in any one family. This latter formula was used in computing the

[44] Boas, Franz. As yet unpublished. I am indebted to Professor Boas for the development of this formula and permission to use it previous to its publication by him.

fraternal variability for the various groupings in this study without computing all the values Boas's original formula would have required.

If the Mendelian hypothesis is to hold, the fraternal variability of the cross between any two racial groups must be somewhat greater than that of either parent group in any trait in which there are decided differences, and the amount of such difference should be greatest at the point of greatest racial mixture. In Table XI the number of families and the fraternal variability by genealogical classes are given, both for the component series and for the entire population.

TABLE XI

Fraternal Variability by Genealogical Classes, All Series

GENEALOGICAL CLASS	ALL SERIES		SERIES I HERSKOVITS		SERIES II KING (HARLEM)		SERIES III KING (W. VA.)		SERIES IV HURSTON	
	No.	S_f	No.	S_f	No.	S_f	No.	S_f	No.	S_f
N (2 parents).....	30	3.36	9	2.90	12	3.41	6	4.05	3	2.73
N (1 parent).....	45	7.12	9	4.43	20	6.02	14	9.61	2	3.40
NNW.....	163	6.19	59	5.65	38	5.93	54	6.76	12	5.92
NW.....	62	6.99	22	6.94	14	7.10	9	8.68	17	4.61
NWW.....	59	7.31	26	6.69	9	12.23	8	6.58	16	4.66
All Classes:.....	309	6.39	125	5.88	93	6.45	91	7.27	50	4.88

The unmixed Negro group is divided into two groups, according as measurements of only one or both parents were available. Where the pigmentation percentage of one of the parents is unknown, there is a chance that he or she may not have belonged to the unmixed Negro group. The greater variability of the *N*(1 parent) group as contrasted to the *N*(2 parents) group indicates that there probably is some white intermixture in the N group with one parent.

Theoretically the fraternal variability should increase in the N, NNW and NW classes, this latter being the most racially heterogeneous, and then decrease with the NWW, which is more nearly white than Negro. This relationship is found in Series I(Herskovits' Harlem), and Series III (King's West Virginia); but in Series II (King's Harlem) the variability of the NWW is very much greater than that of the NW, while in Series IV (Miss Hurston's Selected Harlem), it is slightly greater. Considering the number of families

involved in the NW and the NWW in Series II, 14 in the former and 9 in the latter, it is not felt that much significance may be attached to the increase in variability from 7.10 to 12.23 in this series alone. In the other three series, based on more families, the variability in the NW and the NWW group is approximately the same. The fraternal variability for the entire population shows a steady increase from the unmixed Negro value of 3.36 to the NWW, more White than Negro, value of 7.31.

TABLE XII

Fraternal Variability by Differences in Percentage of N Pigmentation of Parents, by Series

DIFFERENCES BY 6 PER CENT GROUPS	ALL SERIES		SERIES I		SERIES II		SERIES III		SERIES IV	
	No.	S_f	No.	S_f	No.	S_f	No.	S_f	No.	S_f
1–6	96	5.02	37	4.67	23	4.28	17	6.21	19	4.60
7–12	58	6.33	24	6.13	9	9.25	15	4.97	10	2.49
13–18	37	6.16	11	7.50	7	5.90	13	5.68	6	4.17
19–24	15	7.06	3	5.25	7	7.16	3	7.64	2	7.27
25–30	11	7.14	3	5.75	2	7.67	4	6.22	2	8.49
31–36	5	8.09	2	6.69	2	11.35	1	4.73		
37–42	5	7.94	1	4.58	2	8.87	2	9.51		
43–48										
49–54	3	7.69	2	8.52	..	....	1	0.77		
Total.....	230	6.05	83	5.99	52	6.78	56	5.90	39	4.94

Summary, 12 Per Cent Groups

DIFFERENCES OF PARENTS BY 12 PER CENT GROUPS	NO. FAMILIES	S_f, ALL SERIES
1–12	154	5.52
13–24	52	6.43
25–36	16	7.58
37–48	5	7.94
49–60	3	7.69
Total.....	...	6.05

If the phenomena of segregation and recombination characteristic of Mendelism are involved in the inheritance of pigmentation, and if the unmixed Negro and the White represent pure races, then statisti-

cally the variability of an infinite number of offspring of any union should increase with differences in the pigmentation of the parents. To test this hypothesis, the fraternal variability was computed, first for separate series, and then for the combined series, according, first to 3 per cent, then to 6 per cent, and finally to 12 per cent differences in the pigmentation of the parents. As it has previously been shown that there is no difference between the sexes in the matter of N pigmentation, no attention was paid to whether the father or mother in any given family was the darker parent, but only to the difference to the nearest per cent between them. As the observational error of the original measurements is 3 per cent, the variability in steps of 6 per cent (Table XII) gives a clearer picture of the actual relationship between the differences in parental pigmentation and the variability of the offspring. Except for minor deviations, due to the small number of cases, there is an increase in variability of offspring within the family as the difference between the parents increases. If the differences are taken in groups of 12 per cent, the increased variability becomes even more consistent and striking.

Again, if the Mendelian hypothesis is to hold, the variability of the offspring of parents of approximately the same percentage of N pigmentation should increase as the amount of racial heterogeneity of the parents. The Negroes in the middle ranges of N, being theoretically more heterozygous, should produce more variable offspring than either the very light or the very dark. To test this hypothesis, those parents within 6 per cent of the same percentage of N were taken as of the same color. The upper third (67 to 90 per cent) of the entire pigmentation range of 22 to 90 per cent was designated as dark; the middle third (45 per cent to 67 per cent) as medium; and the lower third (22 per cent to 45 per cent) as light. The fraternal variability for these classes as thus determined does not conform to theoretical expectations, but decreases consistently from the lighter to the darker Negro parents. The behavior of the fraternal variability in these cases is very difficult to interpret. There is no bias in the method of pigmentation measurement which could tend to make the variability less in the lower percentages of N than in the higher, but instead the greater accuracy in distinguishing small differences in the per cent of N in the black sector in the lower ranges probably would tend to make the variability less. With the deficiencies of our present data, we cannot offer any tenable theory by which to reconcile the increasing variability from darkest to lightest Negroes

with a Mendelian hypothesis. These data do however show the inapplicability of any theory of blending inheritance in the pigmentation of the American Negro.

TABLE XIII

Fraternal Variability of Offspring of Same Color Parents, by Thirds of Pigmentation Range of the Series

AVERAGE PERCENTAGE N OF PARENTS	NO. OF FAMILIES	S_f
Under 45—light	8	9.43
45 to 67—medium	30	5.54
67 to 90—dark	57	3.84
Total	95	5.04

TABLE XIV

Fraternal Variability of Offspring of Fathers of Varying Percentages of N Pigmentation Mated to Mothers of Varying Percentages

PERCENTAGE N OF FATHER	PERCENTAGE N OF MOTHER	NO. OF FAMILIES	S_f
Under 45	Under 45	5	10.20
	45–67	13	6.45
	67 and over	5	6.49
	Total	23	7.63
45–67	Under 45	11	8.03
	45–67	49	7.16
	67 and over	16	7.00
	Total	76	7.22
67 and over	Under 45	9	7.64
	45–67	48	5.86
	67 and over	72	3.80
	Total	129	5.03
Total, All Types		228	6.06

Various types of matings between the dark, medium, and light groups may be valuable as indicating whether or not Mendelian

inheritance is in operation. Previous analysis would lead us to expect that the offspring of parents who fall into the same class would be less variable for the dark group, and most variable for the light group. If Mendelian dominance of N pigmentation is assumed, then when parents fall into different groups, the variability might be thought to be somewhat less when one parent is in the dark group than if neither is, and less if one is medium rather than both light. Table XIV gives the fraternal variability of offspring for fathers of different groups according to whether the mother was dark, medium, or light. These same data may be rearranged to give the variability of offspring of mothers of different groups according to whether the father was light, medium, or dark. The variability of all offspring of either father or mother becomes greater from the darker to the lighter group, regardless of the pigmentation of the other parent. For any given group father or mother, the fraternal variability becomes greater according to whether the other parent is dark, medium, or light. The greatest variability in all groups is 10.20, where both parents are light; and the least is 3.80, where both are dark. The agreement with general Mendelian theory in this aspect of the results is very close, except that again we should expect the variability of the offspring of medium parents to be greater than that of the offspring of light parents.

Summarizing, the results of a consideration of fraternal variability in the various groups comprising this sample of the American Negro population show that the variability of offspring with the families increases:

1. With larger amounts of white intermixture.
2. With greater differences between the percentages of N pigmentation of the parents.
3. With increasingly smaller percentages of N for parents of the same color.
4. With increasing lightness of the darker parent of the family.
5. And, for any given color parent, with the increasing lightness of the other parent.

The theoretical significance of this behavior of the fraternal variability, taken with the phenomena disclosed by the use of averages and regression lines, is puzzling. Before this point is discussed, it may be of value to consider the variability of the family lines within the general population, and the relation of this variability to the fraternal variability for the same groups.

Family variability may aid in analyzing the process of inheritance, for in a population in which there has been recent crossing, family lines would differ considerably among themselves if segregation in hereditary traits were manifesting itself, while in racially homogeneous populations or those in which there has been long continued intermixture, family lines would all have practically the same ancestry and hence would have a low variability as among themselves.

Since the variability of the total population and of the fraternities within the families has already been computed, the variability of the family lines themselves need not be computed directly, but may be secured by the formula:

$$s = \pm\sqrt{\sigma^2 - S_f^2}$$

where s is the family variability, σ the total variability, and S_f the fraternal variability.[45]

The total, family, and fraternal variability for each genealogical class are given together in Table XV to facilitate comparison. The variability of family lines is in all cases greater than the fraternal variability; that is, the variability between families is greater than the variability within the families themselves. And, just as total and fraternal variability, family variability increases as the amount of white intermixture becomes greater.

TABLE XV

Comparison of Variabilities by Genealogical Classes

GENEALOGICAL CLASS	σ	s	S_f
N	8.93	6.90	5.67
NNW	11.21	9.24	6.19
NW	11.12	8.65	6.99
NWW	12.35	9.95	7.31
Total.....	13.02	11.34	6.39

The results of this comparison of family and inter-family variability for N are strikingly different from those Herskovits secured in five other traits for the Series I (Herskovits' Harlem) alone.[46]

[45] Herskovits, M. J. A Further Discussion of the Variability of Family Strains in the Negro-White Population of New York City. *Jour. of the Amer. Statistical Ass'n*, Sept. 1925, p. 1–10.

[46] *Ibid.*, p. 9.

	s-*Family*	S_f-*Fraternal*
Interpupillary Distance	1.76	±2.35
Width of Nostrils	1.81	±2.10
Height of Ear	2.51	±2.88
Length of Middle Ear	2.81	±4.87
Cephalic Index	1.85	±2.93

How shall we interpret this lack of correspondence in the relative size of family and fraternal variability for these traits and for N pigmentation? That the family variability is actually greater than the fraternal in N pigmentation in this particular sample of the American Negro population is evident from the consistency of the results in each series, each genealogical class, and the total population. Since the difference between these comparative variabilities in pigmentation and the reverse comparative relationship found in Herskovits' Harlem series for other traits does not pertain directly to the subject of this study, only a rather tentative explanation will be made. If family variability is greater than fraternal variability, then, in a population in which the larger amount of racial intermixture took place some generations back, it would seem that the great mass of individuals must be selecting mates with somewhat near, though not necessarily the same as, their own percentage of N pigmentation. If sexual selection as regards pigmentation were non-existent, and mating of Negroes of different shades as haphazard as the throwing of dice, all families would have approximately the same type of ancestry in respect to pigmentation and family variability would be slight. Herskovits' study of social selection in Harlem showed that "the darkest women . . . seem to marry men of about their own color or lighter, but the lighter women, and those not pronouncedly dark, marry men darker than themselves."[47] This produces a curvilinear relationship between the pigmentation of husbands and wives. In the family groups comprising the four series of this study, the average percentage of N of the lighter parent increases as the average percentage of N of the darker parent becomes greater. The regression coefficient of lighter on darker parents, regardless of which sex is the darker and which the lighter, is 2.36 per cent per 3 per cent group width.

The other traits measured by Herskovits are not those on which social selection has come to be based,[48] and hence there would tend to

[47] Herskovits, M. J. Social Selection in a Mixed Population. *Proceedings of the Nat'l Academy of Sciences*, vol. xii (1926), p. 587–593.

[48] Herskovits, M. J. *op. cit.*, p. 593.

be no correlation between husband and wife except that which is due to the correlation in inheritance of these other factors with pigmentation. Doubtless a more thorough analysis of this point would be profitable, but no further digression is pardonable here.

In conclusion, total, family, and fraternal variability show essentially the same tendency to increase as the intermixture of white increases, being least in the N, or unmixed Negro, and usually greatest in the NWW, which is more white than Negro. Family variability is greater than fraternal variability, which indicates that there is greater variability between families of the population than within the families themselves. The known heterogeneity of racial origin and a tendency to selective mating as seen in the regression line of lighter on darker parents when taken together help to explain the relationship of the variabilities to each other. Their significance as indicating the method of inheritance will be discussed later.

POSSIBLE DOMINANCE OF N PIGMENTATION

In any popular discussion, and in many scientific ones, it is either asserted or tacitly assumed, that the greater N pigmentation of the Negro is dominant to the lesser N pigmentation of the white. But whether the offspring tend to approach more nearly to the pigmentation of the darker parent than to the lighter is difficult to analyze when dealing with the heredity of a trait obviously complex and not subject to experimental breeding. If one parent has a greater percentage of N pigmentation than the other, then, assuming an infinite number of children, the average of the children should be nearer the pigmentation of the darker parent than the lighter if N pigmentation is dominant. The only way to test this in human material is to deal with families in the mass. All families were divided into six classes according to whether both parents were dark (over 67 per cent N); one dark and the other medium (45 to 67 per cent N); one dark and one light (under 45 per cent N); both medium; one medium and one light; or both light. The average percentage of N for the fathers, mothers, and offspring of these various types of crosses is given in Table XVI. Here the indications of dominance are slight. For the dark by dark mating, the average percentage of N pigmentation of the offspring is less than that of either parent; for the light by light it is greater; and for the medium by medium almost exactly intermediate between the two parental averages. The dark by medium and the medium by light

give the offspring midway between the average value of the lighter and darker parental averages. The dark by light cross, which is by far the most significant from the standpoint of displaying dominance, gives an offspring much nearer the dark than the light parent, as with the average of the darker parents 75.17 per cent, and the lighter 38.96 per cent, the average of the offspring is 60.99 per cent; 22.03 per cent above the lighter parent and only 14.18 per cent below the darker.

TABLE XVI

Average Percentage of N Pigmentation of Offspring of Various Types of Dark, Medium, and Light Matings

TYPE OF MATINGS	NO. FAM.	AV. PER CENT N DARKER PARENT	AV. PER CENT N LIGHTER PARENT	AV. PER CENT N ALL OFFSPRING	OFFSPRING ± DARKER PARENT	OFFSPRING ± LIGHTER PARENT
Dark × Dark.....	103	79.23	74.43	74.22	− 5.01	− 0.21
Medium × Medium..	70	59.48	52.52	56.01	− 3.47	+ 3.49
Light × Light.....	6	39.20	37.58	43.32	+ 4.12	+ 5.74
Dark × Medium.....	89	74.41	58.77	65.49	− 8.92	+ 6.72
Dark × Light.....	20	75.17	38.96	60.99	−14.18	+22.00
Medium × Light.....	38	54.49	39.82	47.18	− 7.31	+ 7.36

If there is a difference between the pigmentation of the parent, then with dominance, the wider the difference between the parental percentages, the more pronounced should be the resemblance to the darker parent. This is essentially the same method of attack as that pursued by the grouping into dark, medium, and light except that it deals with percentage differences, and not mass classification. All families in which the parents were within 12 per cent of each other's percentage of N pigmentation were grouped as against those in which the differences were over 12 per cent. Treating the average percentage of the lighter parent and offspring in each frequency group of darker parent as equal, regardless of the number of families involved, there is no difference in the average resemblance of offspring to the two parents when the latter are within 12 per cent of each other. Where the difference is over 12 per cent, there is a slightly greater resemblance to the darker parent.

Average of	*Parents within 12 per cent*	*Parents over 12 per cent*
Darker Parents	62.00 per cent N	66.50 per cent N
Lighter Parents	57.16 per cent N	45.33 per cent N
All Offspring	59.53 per cent N	57.26 per cent N
Difference Darker Parent and Offspring	−2.47 per cent N	− 9.24 per cent N
Difference Lighter Parent and Offspring	+2.37 per cent N	+11.93 per cent N

Here is a slight indication of dominance, but since the offspring are only 2.6 per cent closer to the darker parent where the difference is over 12 per cent, and since this is only slightly over 10 per cent of the total difference between the parents, it offers no conclusive evidence except as it may add some slight additional validity to other indications of dominance.

In conclusion, the resemblance to the darker parent is greater than to the lighter if the difference between the parents is over 12 per cent; or if the mating is one of a dark Negro (over 67 per cent N) to a light Negro (under 45 per cent N). All that we are justified in saying is that perhaps pigmentation is dominant in inheritance, but if so, multiple factors must be postulated to explain the very slight evidences obtainable by the various ways in which the data were manipulated to detect greater resemblance to the darker parent.

COMPARATIVE ANALYSIS OF DAVENPORT'S DATA

Since Davenport's original data for the families used in his study are published in his memoir, *The Inheritance of Skin-Color in Negro-White Crosses*, Carnegie Institution of Washington, Publication No. 188, material was available to use as a control on the series of measurements utilized in our own study. Davenport's field workers made measurements by the use of the Bradley Color Top on Jamaica, Bermuda, and Louisiana families, but as only two of these, the Jamaica and the Bermuda, were made by the same field worker, they alone were used for comparative purposes. The families for Davenport's study, unlike our own, were selected as representing certain types of crosses and hence contain much more non-Negroid blood than the general average of the American Negro population. They consist largely of F_1 and F_2 crosses; the American Negroes of today are largely F_3 and later.

Davenport used his data in the uncorrected form, assuming that the ox-blood red corresponded to the haemoglobin of the blood, and

that therefore any addition of the approximately 59 per cent black in the red to the readings of the percentage of black would be invalid. In this study, as explained in the introduction, we have worked on the assumption that the four percentages are quantitative readings which, blended, match the skin, with no implications as to their necessary correspondence to any specific component parts of the skin. Accordingly all Davenport's readings were corrected by subtracting 59 per cent of the red reading for each individual and adding it to the black in order to obtain a figure representing the total black of the blend that matches the skin, and hence comparable in this respect to our own measurements.

The data as thus corrected were analyzed in the same way as our own, the results of which analysis we can only summarize briefly.

TABLE XVII

Average Percentage of N Pigmentation by Age Groups for Males of Davenport's Jamaica and Bermuda Series

AGE	JAMAICA SERIES		BERMUDA SERIES		COMBINED SERIES	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
1	12	44.00	11	44.09	23	44.04
2	6	40.16	10	36.50	16	37.87
3	11	46.82	6	46.66	17	46.76
4	9	39.22	8	42.12	17	40.58
5	9	40.10	8	45.12	17	42.47
6	5	46.80	5	55.60	10	51.20
7	10	48.20	12	40.25	22	43.85
8	12	42.42	6	55.00	18	46.61
9	12	42.10	6	47.00	18	43.72
10	10	45.30	7	52.57	17	48.29
11	3	51.66	7	50.00	10	50.50
12	12	41.43	4	49.50	16	44.36
13	6	41.33	3	45.66	9	42.77
14	8	40.50	2	41.50	10	40.70
15	6	47.00	1	71.00	7	50.42
16	3	30.30	2	53.00	5	39.40
17	5	41.60	1	72.00	6	46.66
18	1	47.00	1	57.00	2	52.00
19	3	38.00	2	59.00	5	46.40
20+	49	37.93	55	45.43	104	41.90

Individuals in Davenport's pedigrees were sorted by series and sexes into age groups (Tables XVII and XVIII). As the precise ages were not given for all adults, all individuals over twenty were put into the general class of adults. Means and standard deviations were computed for the Jamaica and Bermuda Series separately, showing no consistent difference between either sexes or series, so all individuals of an age were combined and the means and standard deviations computed for age groups. The means for these various ages were smoothed by the use of a three-year running average of the means, and an ideal curve drawn to fit the data (Table XIX). There is a steady increase in pigmentation to the age of 9 or 10, and then a steady decline to adulthood. The small number of cases, the selection of specific types of crosses, and the lack of any check on the compara-

TABLE XVIII

Average Percentage of N Pigmentation by Age Groups for Females of Davenport's Jamaica and Bermuda Series

AGE	JAMAICA SERIES		BERMUDA SERIES		COMBINED SERIES	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
1	11	36.00	10	45.20	21	40.38
2	9	42.77	9	42.22	18	42.50
3	11	42.45	3	43.00	14	42.57
4	5	44.20	16	42.25	21	42.71
5	9	46.11	9	48.33	18	47.22
6	10	45.70	7	55.30	17	49.64
7	8	44.50	10	48.80	18	51.83
8	10	52.30	5	51.20	15	53.26
9	6	42.83	6	47.00	12	44.91
10	10	47.70	8	52.63	18	49.88
11	6	42.66	6	53.00	12	47.83
12	13	44.61	8	53.00	21	47.80
13	8	50.00	5	52.00	13	50.76
14	7	48.71	7	49.57	14	49.14
15	8	44.25	3	54.66	11	47.09
16	6	45.66	5	52.40	11	48.72
17	4	43.00	1	50.00	5	44.40
18	6	38.83	5	51.40	11	44.55
19	1	51.00	..	.....	1	51.00
20+	116	46.12	93	49.54	209	47.64

bility of the results of the individual measurers, make the growth curves for Davenport's series and our own non-combinable.

TABLE XIX

Age Changes in Percentage of N Pigmentation of the Negro, Davenport's Data Smoothed and Idealized for Both Sexes and All Series

AGE	NUMBER INDIVIDUALS	ACTUAL DATA PER CENT N	SMOOTHED PER CENT N	IDEALIZED PER CENT N
1	44	42.29	41.74	41.50
2	34	40.32	42.41	42.00
3	31	44.87	42.22	43.00
4	38	41.76	43.75	44.00
5	35	44.91	45.15	45.00
6	27	52.20	47.31	46.00
7	40	47.45	48.72	47.00
8	33	49.03	47.00	47.75
9	30	44.20	47.58	48.00
10	35	49.11	47.40	47.75
11	22	49.04	48.20	47.67
12	37	46.62	47.57	47.50
13	22	47.50	46.56	47.25
14	24	45.62	47.04	47.00
15	18	48.38	46.53	46.87
16	16	45.81	46.80	46.75
17	11	45.63	45.72	46.50
18	13	45.68	45.96	46.37
19	6	47.16	46.40	46.25
20+	313	45.73	45.73	46.00

After correction of all individual readings for age changes, the frequency distribution of neither the Bermuda, the Jamaica, nor the combined series show any significant traces of modality (Table XX). The peaks in the Bermuda polygon which Davenport found at (1) about 5 per cent; (2) about 18 per cent; (3) about 35 per cent; (4) about 46 per cent; and (5) about 70 per cent have disappeared, and whatever slight irregularities remain are probably accidental and due to paucity of data.

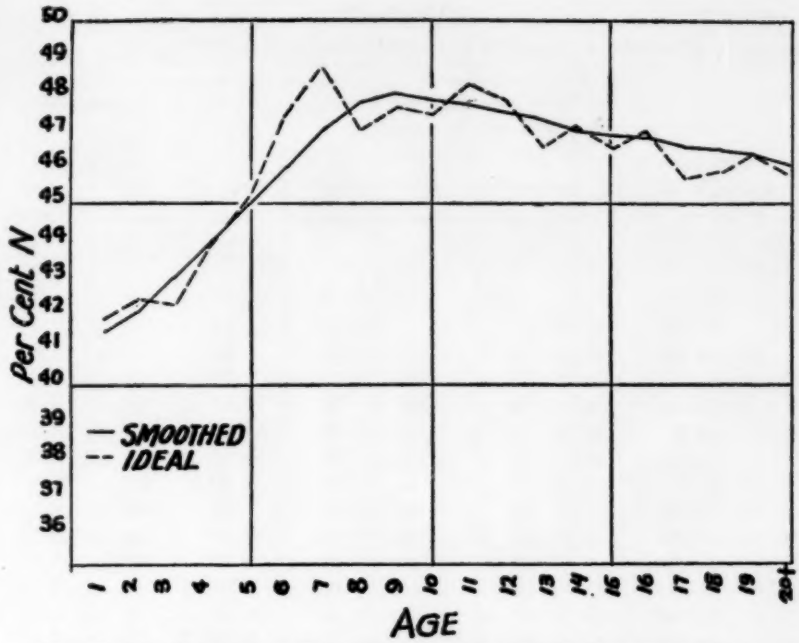

FIG. 5. AGE CHANGES IN PIGMENTATION, DAVENPORT'S COMBINED SERIES

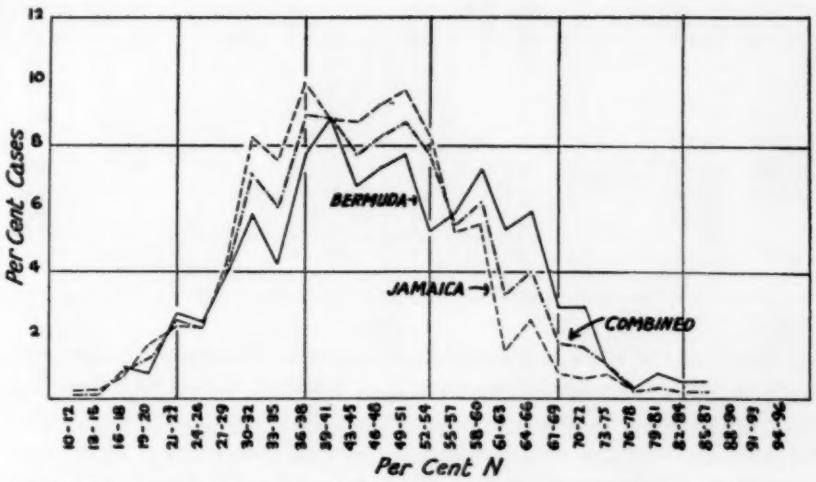

FIG. 6. PERCENTAGE FREQUENCY DISTRIBUTION OF DAVENPORT'S JAMAICA-BERMUDA SERIES, BY PER CENT OF N PIGMENTATION

TABLE XX

Frequency Distribution of Davenport's Series, Corrected Data

PER CENT N	BERMUDA		JAMAICA		COMBINED	
	No. Cases	Per Cent	No. Cases	Per Cent	No. Cases	Per Cent
10–22	....	....	1	.22	1	.12
13–15	....	....	1	.22	1	.12
16–18	4	1.07	3	.67	7	.85
19–21	3	.80	8	1.78	11	1.34
22–24	10	2.68	11	2.44	21	2.55
25–27	9	2.41	10	2.22	19	2.31
28–30	15	4.02	20	4.44	35	4.25
31–33	22	5.90	37	8.22	59	7.17
34–36	16	4.29	34	7.55	50	6.08
37–39	29	7.77	45	10.00	74	8.99
40–42	33	8.85	40	8.89	73	8.87
43–45	25	6.70	39	8.67	64	7.78
46–48	27	7.24	42	9.33	69	8.38
49–51	29	7.77	44	9.78	73	8.87
52–54	26	6.97	37	8.22	63	7.65
55–57	22	5.90	23	5.11	45	5.47
58–60	27	7.24	25	5.56	52	6.32
61–63	20	5.36	7	1.56	27	3.29
64–66	22	5.90	11	2.44	33	4.01
67–69	11	2.95	4	.89	15	1.82
70–72	11	2.95	3	.67	14	1.70
73–75	4	1.07	4	.89	8	.97
76–78	1	.27	1	.22	2	.24
79–81	3	.80	..	....	3	.36
82–84	2	.54	..	....	2	.24
85–87	2	.54	..	....	2	.24
Total.....	373		450		823	

To make the study comparable to our own, Davenport's data were used in family groups of parents and children only. The average percentage of N pigmentation for sons, daughters, and all children for parents, fathers, and mothers of varying percentages of N show the same fundamental relationship as our own American Negro series.

TABLE XXI

Average Percentage of N Pigmentation of Children of Parents, Fathers, and Mothers of Varying Average Percentages of N, Davenport's Combined Series

AVERAGE OF	AVERAGE OF OFFSPRING FOR					
	1. Parents		2. Fathers		3. Mothers	
	No.*	Per Cent	No.	Per Cent	No.	Per Cent
10–12	..	.....	1	52.25	..	.....
13–15	..	.....	1	45.89	..	.....
16–18	..	.....	2	39.96	1	38.50
19–21	..	.....	2	43.40		.....
22–24	1	26.75	10	38.65	4	35.14
25–27	2	38.15	1	53.41	6	32.15
28–30	2	39.54	4	48.02	3	38.49
31–33	7	38.98	7	44.85	8	39.42
34–36	7	39.40	6	44.84	10	39.58
37–39	7	40.79	6	45.30	13	41.33
40–42	9	45.19	4	43.56	16	39.21
43–45	12	47.39	9	45.85	11	43.66
46–48	3	50.44	7	50.74	10	46.84
49–51	2	59.47	7	52.78	10	42.75
52–54	5	56.81	6	50.69	17	51.01
55–57	3	53.77	3	54.54	9	50.07
58–60	7	57.34	2	61.88	15	55.75
61–63	4	65.26	1	65.88	4	48.34
64–66	..	.....	1	68.70	11	56.78
67–69	..	.....	..	.....	8	56.00
70–72	1	69.00	..	.....	1	63.37
73–75	..	.....	..	.....	2	60.50
76–78	..	.....	1	49.00	1	49.54
79–81	..	.....	..	.....	1	64.43
82–84	..	.....	1	69.00	1	55.08
85–87	..	.....	..	.....	2	55.68
Regression (3 per cent Groups).....	..	2.41	..	1.04	..	1.69

* *Number* refers to number of families.

The regression coefficients for the two series, Herskovits' and Davenport's, may be compared directly in Table XXII. Considering the fact that the original measurements of the two series are by different individuals, with no possible check on the comparability of the

two series; that our series is of American Negroes, nonselected, and his West Indian, selected; and that our series represents a much larger number of cases, the similarity of the average and the regression coefficients for the two series is surprising.

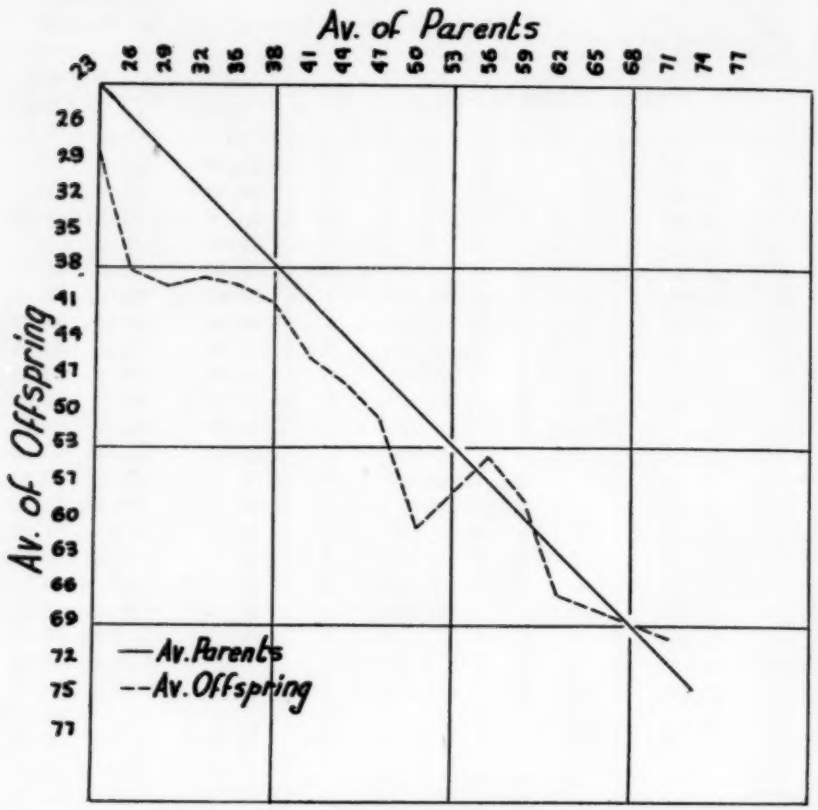

FIG. 7. REGRESSION OF OFFSPRING ON MEAN PERCENTAGE OF N PIGMENTATION OF PARENTS, DAVENPORT'S COMBINED SERIES

All the various groupings for which the fraternal variability was computed in our own series could not be made with Davenport's data, owing mainly to the fact that the number of cases was not great enough to make any results significant. However, the fraternal variability for percentage differences in N pigmentation of the parents in groups of 12 per cent shows an increase as the difference between the parents becomes greater, with a slight decrease when the difference is from 25 to

TABLE XXII

Comparison of Regression Coefficients (Herskovits') American Negro and (Davenport's) West Indian Series

REGRESSION ON	ALL OFFSPRING		SONS		DAUGHTERS	
	Am. Negro	Dav. Negro	Am. Negro	Dav. Negro	Am. Negro	Dav. Negro
Parents	2.43	2.41	2.78	2.74	2.59	2.14
Fathers	1.48	1.04	1.43	1.72	1.45	1.21
Mothers	1.84	1.69	1.77	.93	2.81	1.20

36 per cent, but the small number of cases, only 13, makes it probable that this result is entirely accidental. The fraternal variability is greater in every case for Davenport's data than for our own, probably owing to the fact that he used selected Negro-White crosses, while our own data, being nonselected, are the result of less intermixture, and most of that several generations removed.

TABLE XXIII

Comparison of Fraternal Variability by Differences in Percentage of N Pigmentation of Father and Mother, Herskovits' and Davenport's Series

DIFFERENCE IN PER CENT N OF PARENTS	HERSKOVITS' SERIES		DAVENPORT'S SERIES	
	No. Fam.	S_f	No. Fam.	S_f
1–12	154	5.52	33	8.01
13–24	52	6.43	18	9.84
25–36	16	7.58	13	9.26
37–48	5	7.94	..	....
49–60	3	7.69	..	....

When all parents of approximately the same color are divided into dark, medium, and light according to thirds of the pigmentation range of Davenport's series, the fraternal variability increases from the dark to the light classes. Table XXIV compares Davenport's and Herskovits' series as to relative variability of the dark, medium, and light groups in their own series. In both cases, the offspring of light parents of the series are more variable than those of dark of the same series, though dark for Davenport's series is medium for Herskovits'.

Hence in Table XXV the actual percentage groupings for the two series are made comparable rather than the relative portions of the separate series. Here again the similarity is remarkable when it is remembered that Davenport's is a selected series, largely F_1 and F_2. Either this fact, or the small number of cases, may account for the slightly greater variability in all groups of Davenport's series as compared with Herskovits'.

In summary, when Davenport's series are corrected for the percentage of black in the ox-blood red sector, and for age changes, and analyzed by family groups:

1. There is no bimodality (or other than mono-modality) in the frequency distribution of either sex considered separately, or the combined sexes.

TABLE XXIV

Comparison of Fraternal Variability of Offspring of Dark, Medium, and Light Parents, Herskovits' and Davenport's Series

HERSKOVITS' SERIES			DAVENPORT'S SERIES		
Av. Per Cent N Parents	No. Fam.	S_f	Av. Per Cent N Parents	No. Fam.	S_f
Dark—Over 67	57	3.84	Dark—Over 44	6	6.56
Medium—45–67	30	5.54	Medium—36–44	5	7.67
Light—Under 45	8	9.43	Light—Under 36	6	13.32

TABLE XXV

Comparison of Fraternal Variability of Offspring of Dark, Medium, and Light Parents by Percentage Groupings

AV. PER CENT N PARENTS	HERSKOVITS' SERIES		DAVENPORT'S SERIES	
	Class	S_f	Class	S_f
Under 45 per cent N	Light	9.43	Light	13.32
			Medium	7.67
45 per cent N and over	Medium	5.54	Dark	6.56
	Dark	3.84		

2. There is a regression of the offspring on the average percentage N of parents of 2.43; on fathers, 1.04; and on mothers, 1.69.

3. Fraternal Variability increases with increasing differences in the pigmentation of the parents, and, when the parents are of approximately the same color, with less percentages of N.

Davenport's data are thus in substantial agreement with our own and subject to the same interpretation.

THEORETICAL SIGNIFICANCE OF THE ANALYSIS

Thus far the facts have been presented with almost no attempt at an analysis of their theoretical significance in regard to the hereditary mechanisms involved in producing the type of resemblance of parents and offspring which has been shown to exist.

The negative eliminations are far easier than the positive analysis. The N, which may conceivably represent the amount of melanin pigmentation of the American Negro, is not inherited as a blend, using the word in the sense of a fusion of hereditary characteristics without a segregation in later generations. Both the regression coefficients and the fraternal variability make this explanation tenuous. If there were a blend, the variability of the offspring of parents of approximately the same percentage of N would tend to be very slight, and what variability there was, due to environmental influence, would be practically the same regardless of the comparative lightness or darkness of the parents. The fact that the variability of the offspring of parents of the same percentage of N pigmentation increases with the increasing lightness of the parents is irreconcilable with any theory of blending inheritance yet the average of the children of parents in the middle ranges of pigmentation, from about 50 per cent N to 69 per cent N, is usually within the same 3 per cent group as the average of the pigmentation of their father and mother. The problem is to account for this appearance of a blend which cannot be a blend if the regression coefficients and expressions of fraternal variability are valid.

Neither is pigmentation inherited according to the simple one-factor Mendelian mechanism. Assuming there were a factor for N pigmentation which behaved as a Mendelian dominant, then there should be concentration of numbers at some percentage of N representing the dominant NN, the heterozygote Nn, and the recessive nn, with many more cases of the dominant, the precise number depending on the amount and type of intermixture which has occurred. But if we examine the frequency distribution, there is in all series and in the

combined series a monomodal curve, with concentration of numbers in the upper ranges. Again, how could we account for the fact that there is consistency in the change of the average pigmentation of children with a 3 per cent frequency distribution of the increasing average pigmentation of their parents, their fathers, or their mothers, if the germ class of an individual could only be of the combination NN, Nn, and nn, where N represents the factor for N pigmentation, and n either the recessive factor or the absence of the dominant N? And if the frequency groups in the middle ranges represent the Nn heterozygote, with N only partially dominant, then how shall we explain the fact that the average of the offspring coincides so nearly with the average of the parents? If two Nn marry, then the expectation would be 1NN, 2Nn, and 1nn, which, though when averaged might give the appearance of an entire Nn population, certainly could not have a lower variability than the offspring of two individuals, both of whom have a small percentage of N pigmentation, and hence theoretically are nn and so can have only nn offspring, or perhaps are Nn and nn, and so have ½Nn and ½nn offspring? This procedure might be continued indefinitely, showing the futility of the application of a one factor Mendelian explanation to our data. Simplicity of explanation is perhaps desirable when possible in scientific work, but obviously here we have a complex phenomenon, and one not resolvable into a black × white equals black which when mated with black *inter se* produces 3 black and 1 white, no matter how much the underlying phenomena may be obscured or modified by environmental conditions.

Practically the same objections may be urged against the two factor Mendelian explanation as against the one factor. It is too simple an explanation to fit the phenomena when all facts are taken into account. There is no trace of concentration in the frequency distribution at certain percentages which might correspond roughly to the individuals having the various genetic compositions. Davenport found such concentration in his Bermuda series, but when his own data were corrected for the black in the red and for age changes, this concentration disappeared. To repeat, all the curves, for all series and for all genealogical classes, are essentially monomodal. Two factors can no more account for the consistency of change between parents and offspring, or fathers, and mothers and children, than can the one factor hypothesis, unless we posit a very great environmental influence on pigmentation which obscures the distribution which the genetic composition of the individuals would otherwise produce. But if we posit

this, then we are admitting the invalidity of any study of the heredity of pigmentation which is based on measurements of the color of individuals. The consistency of the differences between genealogical classes in all types of manipulation of the data argues against sufficient environmental influence, either external or internal to the individual, to obscure the working of whatever hereditary mechanisms are involved. Again, in a two factor hypothesis, how could one explain the fact that the variability of the offspring of two light parents is greater than that of the offspring of parents in the medium range whose genetic composition must be more heterogeneous? The two factor Mendelian hypothesis is insufficient to explain all the facts.

Can one posit Mendelian heredity, with its independence of genes or factors, their segregation and later recombination, in any form as holding for the inheritance of pigmentation in the American Negro? What are the criteria by which Mendelian inheritance in any population may be recognized, disregarding for the moment the question of the precise analysis of the number and type of factors involved, and their mutual inter-relationships? If two races diverse for any particular trait inter-breed, then the hybrid offspring must show a greater variability in regard to that trait than either parent race. Theoretically the point of greatest racial heterogeneity should be the most variable in respect to any trait in which there are decided differences as between the races. Does this criterion of Mendelian inheritance hold for the N pigmentation of the American Negro? Both fraternal and family variability increase as the amount of white intermixture increases, but the point of greatest variability is in most cases the group nearest the white race and not the one of the greatest racial heterogeneity. In the fraternal variability by series we find the following results, which offer many complications difficult to interpret. In Series I, II, and III, the variability in the NW group is greater than in the NNW, which conforms to expectations; but in the NW in Series IV, the variability is less than in the NNW, which is nearer the Negro race. However, since Series IV has so few families in both the NNW and the NW, no great importance may be attached to this slight point. In Series I and III the variability is greater at the point of greatest racial mixture, the NW, and less in the NWW. In Series IV the NW and the NWW are practically the same, while in Series II there is great discrepancy. This series, it will be remembered, was measured by King and corrected to make it comparable with the measurements of Herskovits. Considering the possible

inaccuracies in the genealogies, and the fact that the dual correction for age changes and the personal factor in measurement may have introduced some errors, Series I, to which the others were corrected, may give a more valid picture than the combined series. If it does, then Mendelian expectations are fulfilled.

Fraternal Variability

Genealogical Class	*All Series*	*Series I*	*II*	*III*	*IV*
N (Both Parents)	3.36	2.90	3.41	4.05	2.73
N (One Parent)	7.12	4.43	6.02	9.61	3.45
NNW	6.19	5.65	5.93	6.76	5.92
NW	6.99	6.94	7.10	8.68	4.61
NWW	7.31	6.69	12.23	6.58	4.66

But when one turns to the total variability of the various series, it is in Series I and II greater in the NWW than in the NW; and in Series III and IV less. In Series II and IV, it is greater in the NNW than in the NW, and in Series I and III less. In the combined series, total variability, except for a slight decline in the NW, increases as the increase of white inter-mixture. The trend here, as in fraternal variability, appears to be a consistent increase with increasing admixtures of white, rather than with increasing racial heterogeneity.

Total Variability

Genealogical Class	*All Series*	*Series I*	*II*	*III*	*IV*
N	8.93	5.50	7.85	11.51	4.63
NNW	11.21	8.98	12.39	10.49	10.41
NW	11.12	9.88	12.32	12.37	8.91
NWW	12.35	10.78	14.60	11.89	7.62
Total	13.02	11.61	13.33	12.75	10.91

In the division of the total population into groups of dark, medium, and light, the latter, while probably nearer the white group and thus representing less racial mixture than the medium, is in all cases the most variable. The combined series indicate greater variability in both the NWW and the NNW than in the NW, the most racially heterogeneous. Even granting that there might theoretically be an increase of variability with dominance of N, the difficulty of reconciling the behavior of these variabilities with Mendelian theory is very great.

But if there is Mendelian inheritance, the variability of the offspring should increase with increasing differences in the pigmentation

of the parents. This is undoubtedly true of our data, the increase being persistent if differences are taken in 12 per cent groups.

If the parents are of the same percentage of N pigmentation, the variability of the offspring of parents of the higher percentages should be least, those of the medium greatest, and those of the light less again. In our data, the variability of the offspring of dark parents is least, that of the medium group greater, but that of the light group greatest. This greater variability of the offspring of lighter parents, nearer the white race, and hence theoretically both homozygous for more recessives and probably heterozygous for more of the dominants, is an enigma similar to the greater fraternal variability in the NWW in the majority of cases.

Thus there are some indications that the inheritance of pigmentation does follow those mechanisms we call Mendelian. Yet can Mendelism explain the regression found for the average values? Here it must be remembered that we are dealing with means, either of parents or children as the case may be, and that individual variations are hidden or obscured. A family in which there are two children of 60 per cent N will give the same average figure for offspring as if the children were 40 and 80 per cent N respectively. When the average of the parents is from 45 to 67 per cent N, the average percentage of N of the offspring generally falls within the same 3 per cent group as that of their parents, and yet the fraternal variability of the offspring of parents within this same general percentage group is 5.54. When the average percentage of N of the parents is high, say over 67 per cent, the average of the offspring is less than that of the parents, and the fraternal variability only 3.84; where the average of the parents is low, say under 45 per cent, the average of the offspring is higher than that of their parents, and the fraternal variability 9.43.

When we pierce the regression, based on mean percentages, of any given group of offspring on parents, we find the variability to conform somewhat to Mendelian expectations. Can we assume that quantitative Mendelian heredity is in operation and explain the phenomena of regression? In an attempt to do so, it is necessary to enter far into the realm of theory. Suppose we assume that pigmentation is due to several factors which behave as Mendelian dominants. Most of those individuals who have high percentages of N pigmentation will be heterozygous for one or more of the factors for N. When two individuals mated who, though outwardly dark, are heterozygous for the factors involved, their offspring would have a

smaller percentage of N pigmentation on the average than the mean pigmentation of their parents. The difficulty practical breeders face in "breeding up" for any particular trait in the production of which multiple factors are involved, illustrates the same type of phenomena. On the other hand, when the average of the parents in under 45 per cent N, one parent is usually darker than the other, as was shown by Herskovits in his analysis of social selection in the American Negro population. Hence on an assumption of Mendelian dominance of the factors for N, the average of the offspring would be a higher percentage of N than the average of the parents. Thus, given freedom to make a sufficient number of subsidiary postulates, Mendelism could explain the averages of children for various percentages of N of the average of the parents. The behavior of the averages of offspring for various percentages of fathers and mothers taken separately can be made to appear Mendelian by the same type of logic. Since very dark individuals marry persons somewhat lighter than themselves, and very light individuals persons somewhat or much darker, then if pigmentation is produced by one or more Mendelian dominants, the offspring of dark fathers or mothers would tend to be lighter, and those of light fathers or mothers darker. As would be expected under this hypothesis, the regression coefficient on either fathers or mothers is less than the coefficient on the mean of the parents. However, the validity of this extremely tenuous type of logic is very questionable. Given the liberty to make a sufficient number of assumptions, a skillful logician could make the results we have secured conform to practically any theory of heredity.

The fact that the regression is greatest for the unmixed Negro group, and becomes less for the NNW and NW is in accord with Mendelian expectations, as the offspring of any racial group resemble their parents more than do the offspring of parents who, representing racial mixture, are heterozygous for the factors involved in many cases. But how shall we explain the fact that the NWW, a group racially more homogeneous than the NW, has a less coefficient of regression?

Perhaps the N pigmentation of the American Negro is a case of Mendelian inheritance, but, if so, it is a complex kind which defies analysis in a human population. Popular *mores* and the necessary length of time involved prohibit controlled breeding experiments which might give results at all comparable with those obtained in the lower plants and animals. That variability increases progressively as the intermixture with the white race shows either that we are dealing with a

faulty technique of pigmentation measurement and biometrical analysis, or that there is here something in inheritance which no present theory of inheritance can explain on the basis of the data which we have available. If it be multiple factor Mendelism, then we have as yet no perfected technique for analyzing either the number of factors involved or their behavior in relation to each other in a human population. We have arrived at an impasse where only deductive logic or hypotheses which defy verification by our present methodology remain.

GENERAL CONCLUSIONS

The general conclusions of this study of the method of inheritance of the pigmentation of the American Negro may be stated as follows:

1. There are no sex differences in N pigmentation.

2. The percentage of N pigmentation of the American Negro increases quite rapidly until puberty, with a maximum at the age of 15; decreases rapidly until about the age of 35; and then decreases very slowly the remainder of life.

3. The frequency distribution of all series, and for each genealogical class, is monomodal, with no concentration at points which might indicate Mendelian genetic combinations.

4. The average percentage of N pigmentation of offspring shows regression to the mean pigmentation of the group, the regression coefficient of offspring on parents (in 3 per cent groups) being 2.43; on fathers, 1.48; and on mothers, 1.84.

5. Both total variability, and its component family and fraternal variabilities, tend in general to increase with increasing intermixture with the white race, being greatest in the group genealogically nearest the white race instead of in the group racially most heterogeneous.

6. Fraternal variability increases with increasing differences between the percentages of N pigmentation of the parents.

7. Fraternal variability of the offspring of parents of the same percentage of N pigmentation is greater the less the percentage N of the parents.

8. Offspring resemble the parent with the greater percentage of N (the darker) slightly more than the one with the less percentage of N (the lighter).

9. Pigmentation is not found on analysis to be inherited by blending, nor is it produced by the action of one or two factors which act as Mendelian dominants. Whether it may be interpreted through

the hypothesis of multiple factors, and, if so, how many factors may be postulated, is not subject to determination from our data by any technique which we know at present.

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PRIMOGENITURE AND DEVELOPMENTAL ANOMALIES

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There is a common tradition that the first-born of the family is more apt to exhibit hereditary defects than are the subsequent children. Nor are hereditary defects the only ones considered to have a predilection for the eldest; so-called congenital malformations, usually regarded as non-hereditary, are supposed to affect the first offspring with undue frequency. Thus it is common to see the statement that congenital hypertrophic pyloric stenosis, an example of the second group of cases, is found in the first child of the family more often than it should be were it apt to affect all members of the family equally. Other anomalies of development which are supposed to show a special tendency to affect the first-born are the various types of congenital heart defects. As to the hereditary group of cases, any disease which is inherited, such as hemophilia, night blindness, polydactylism, etc., is looked upon by many as the especial birth-right of the eldest. If this tradition of a greater susceptibility of the first-born to developmental defects, hereditary or otherwise, be true, it has a marked eugenic significance. If it is not well founded, its acceptance should be challenged, and its implications proved false, not only for the eugenic application involved, but also for the sake of establishing scientific truth. The present paper is an attempt to interpret correctly the statistics which centre about this question.

This idea may have gained some support from experience with domestic animals, for it has been claimed that the first litters sometimes contain fewer members, and also a higher percentage of underdeveloped members, than do the later litters. This, if true, is readily comprehensible; since animals, in nature, became pregnant as soon as sexual maturity is reached; a state that may be attained long before

the female has become fully grown. Hence the young of such half grown animals would tend to be smaller, and fewer in number, inasmuch as there would not be as much room in the uterine cavity as there would be when the animal had reached its full development. This condition is rarely met with in the human female in civilized communities, who in the vast majority of cases has come to the age of physical as well as sexual maturity before the first pregnancy begins.

For clarity in exposition of a problem which of necessity is mathematical in its treatment, the conclusions which have been reached by a critical study of the literature will be set forth at the beginning, and the argument presented later, even as one does in a theorem in geometry. Because opposite conclusions have been reached with respect to some of the points by Still (nor is he alone in his interpretation of the data), his deductions and his method of arriving at them will be taken up in detail through the paper.

The propositions for which I intend to present evidence are as follows: (1) The first-born is *not* more liable to show hereditary defects than are the other children. (2) The first-born is not more liable to show so-called congenital malformations, (usually regarded as non-hereditary), than are the subsequent offspring. This group will be referred to for the sake of brevity, as the "Congenital Group." (3) These congenital defects, looked upon as due to mistakes on the part of nature in preparing suitable environment for the fetus rather than to inherent defects in the fertilized ovum, are in reality inherited and should be classed with the hereditary cases.

The hereditary and congenital groups will however be considered separately for the following reason: If the conception which is fundamental to the science of heredity, namely the free assortment of the chromosomes, be true, then it is chance alone which rules the destiny of the offspring, and chance alone which allots the defect among the members of the family. Hence the first-born, from the theoretical standpoint, should be no more liable to hereditary defects than are any of the other children. If however the congenital cases do show a predilection for the eldest, such tendency would be obscured by the grouping of the hereditary cases with them, especially if the hereditary cases were in the majority. Thus to give the protagonists of the theory that primogeniture is an aetiological factor in the production of malformations the full benefit of the doubt, the two groups will be treated separately.

METHOD OF SOLVING THE PROBLEM

Because the problem presents difficulties in its solution, the methods of attack will be briefly outlined, and their disadvantages mentioned. If all the families in which a malformed child occurred were complete, that is, if all the children who were to be born, were born at the time the statistics were collected, the matter would be easily solved. But because the families are presumably still capable of increase when the physician makes his report of the malformed infant, it is impossible to say whether more children will be born to that mother and whether they will be malformed or normal. It is this "Incompleteness" of the family which is at the basis of the difficulty in solving the question.

Were the families complete, it would be simple enough to compute the proportion of the first children which were defective, the proportion of the second and third children who were deformed, and if all the percentages were about equal, then there would be no special liability on the part of any one child to show the defects. Of course it might be an abnormality which affected only five per cent of the children in each place in the family, or it might be one which affected seventy-five per cent, but provided it affected all equally, it would not matter. Another way of dealing with the problem would be as follows: Compute the percentages of the first-born, etc., among the total number of children, which values if plotted in a curve would give a gradually falling line as one went from the first to the last child. This is the "standard curve." It would resemble the curve seen in figure 1. Then compute the percentage of affected first- and second- and third-born in the total number of *affected* children, and plot the curve for these values, designating it as the "affected curve." If there is no special liability on the part of any one child to show the defects, the curves should be parallel, and should be capable of being placed one over the other with very little variation between them.

When we are dealing with families which may be incomplete what are we to do, since obviously any percentages computed today may be upset tomorrow upon the advent of another infant, whether normal or malformed? If we could be sure that when once a malformed infant has been born into the family, there would not be a second defective one in the same family, we could use as the standard curve, one based upon the population at large; thus assuming that these families, when complete, would have about as many children as

the average family. The affected curve would be based of course upon the number of affected children in the families we were actually dealing with. These curves should be capable of being fitted one over the other, without there being any marked deviations, provided that there is no special liability on the part of any child to exhibit the malformations. A marked liability on the part of any one child would be shown by a wide divergence of the curves at that point.

But the great difficulty is that we cannot be sure that no more malformed children will be born in these families, and alter the "affected curve." Hence the method of choice is that referred to first in the foregoing paragraph, namely, the determination of the proportion of affected children *in each place in the family*. Here the first value cannot be changed by birth of more children, since every family to be listed in this group must have had their first child. The birth of other children in the families would alter the other percentages, either up or down, depending upon the presence or absence of defects. For example, if we found that in a group of one hundred families, there were 20 per cent of the one-hundred first-born affected, and 15 per cent of the sixty second-born affected, then the birth of ten more second children would alter the value, 15 per cent. If they were all normal we should have nine out of seventy children, or practically 13 per cent, affected, but if they were all ten abnormal, we should have nineteen out of seventy affected or 27 per cent. Thus birth of subsequent children could not alter the value in the first place but could either raise or lower that in the following places, so that this method is not without its drawbacks. It has the advantage however over the comparison of affected with standard curves, that one is dealing here with only the members of the affected families, and is not comparing them with some other families, who may have more or less offspring than those under consideration. Both methods were tried in dealing with the two groups of cases in this paper, but in each instance, the standard curve was based upon the families from which the affected persons came, rather than upon some other set of families.

HEREDITARY CASES

The group of hereditary cases will be dealt with first, since theoretically we shall expect no greater liability on the part of the first-born toward them. A series of pedigrees was selected in which the order in the family of the affected and unaffected members was given. The

families were for the most part complete, being in pedigrees extending over several generations. The defects included peroneal atrophy, night blindness, hemophilia, ankylosis of the finger joints, amaurotic family idiocy, multiple cartilaginous exostoses, myotonia congenita, sickle cell anemia, retinitis pigmentosa, cataract, hemorrhagic telangiectasia, Mongolism and chronic hemolytic jaundice.

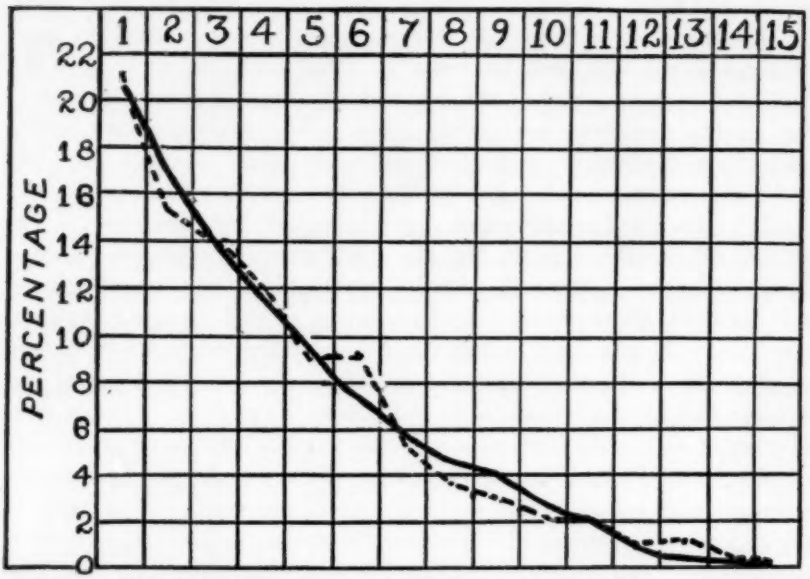

FIG. 1. SOLID LINE REPRESENTS THE PERCENTAGES IN EACH PLACE IN THE FAMILY OF 1800 PERSONS, IN 370 FAMILIES, IN WHICH THERE WERE 600 PERSONS AFFECTED WITH HEREDITARY DEFECTS. BROKEN LINE REPRESENTS THE PERCENTAGES OF THESE AFFECTED 600 OCCURRING IN EACH PLACE IN THE FAMILY

There were in this group 370 families with a total of 1800 persons, of whom 600 were affected. The standard curve was based upon the 370 families, and is shown in figure 1., solid line. This agrees very closely with the standard curve shown in Still's paper. The broken line in figure 1 illustrates the percentage of these 600 cases which were first or second-born in the family. The two curves fall very close to each other, showing that there was not any greater tendency for the first child to be affected than there was for any other member of the family.

Let us now examine the percentages of first, second and third-born which are affected, as plotted in figure 2. This differs from the dotted line in figure 1 in this way. Figure 1 represents the percentage of the

600 cases which were first or second-born, etc., in the family; but figure 2 shows what percentage of the 370 first-born in these families were defective, what proportion of the 297 second-born were malformed, etc. Here we see that of the first-born there were 35.3 per cent, of the second-born, 33.3 per cent and of the third-born 34 per cent affected. The curve in no way resembles that in figure 1, but appears to be a series of values fluctuating on either side a straight line, which would be the average for all the percentages there listed. The probabilities are that were the number of cases to be increased, the deviations on either side the average, would disappear, and the curve approximate more nearly to a straight line.

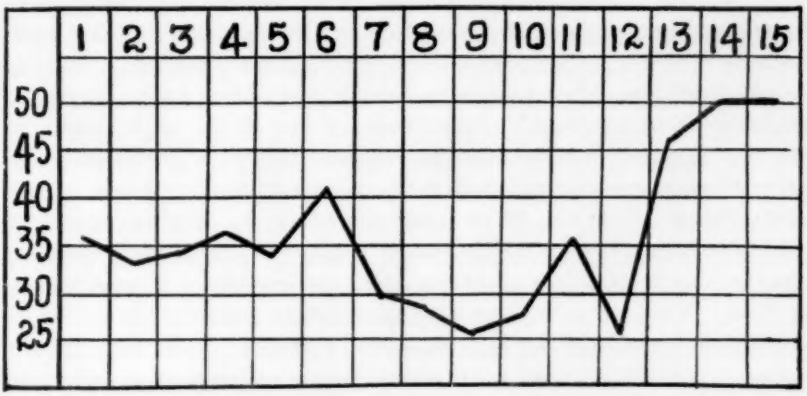

FIG. 2. REPRESENTS THE RATIO OF AFFECTED TO UNAFFECTED MEMBERS IN EACH PLACE IN FAMILY IN THE 370 FAMILIES ABOVE

Another way of checking the accuracy of our conclusions is to determine the average number of pregnancies in the 370 families here listed, and to compute the average pregnancy which resulted in a deformed child. If there is an undue tendency for the first to be affected, this will cause the latter figure to be displaced nearer to the valued one, than is the case with the former average. If however the two values coincide, this will indicate that there is no undue tendency for the first child to be affected.

This calculation was made, and the probable error determined. In these 370 families the average number of pregnancies was found to be 4.15 ± 0.08. The average pregnancy which produced the defective child was 4.18 ± 0.02. The pregnancy which resulted in the defective child was thus found to be slightly higher than the average number of pregnancies in these families, namely, $4.18 - 4.15$, or 0.03 ± 0.08.

Thus the difference between these averages is less than the probable error for that difference, and indicates the practical identity of the curves.

CONGENITAL CASES

Let us now examine those congenital cases which are usually regarded as non-hereditary, and due to some mistake in development, or as Still believed, due in large part to inexperience of the uterus. He advances the idea that Nature makes mistakes, and she is more apt to make them at the first trial than in later ones, hence the greater liability of the first-born to defects. Since the function of the uterus is to provide protection and nutriment for the fertilized ovum, any inexperience it might show would be in the direction of inadequate housing facilities, or deficient blood supply. Either of these conditions would result in delayed implantation of the ovum with consequent impairment of nutrition. Now Stockard has shown with respect to the fish egg that if the nutrition, oxygen supply, or temperature be lowered past the physiological needs of the developing ovum at certain critical moments in its embryonic history, malformations of various types will result, the site of the defect depending upon the time in the developmental period that the inhibition to growth was applied. Thus by choosing the appropriate moment in which to retard the growth of the egg, all sorts of abnormalities may be produced. Individuals may exhibit duplication of parts or of the entire body; the twins may be separate or joined throughout greater or lesser extent of their anatomy. The defect may be one of suppression rather than duplication, so that the mature form may be lacking in any of its organs either in whole or in part. For this reason, such congenital abnormalities as anencephaly, spina bifida, meningocoele, ectopia cordis, ectopia viscerae, atresia of the digestive tract, diaphragmatic hernia, cyclops, etc., were chosen. These should give evidence of the part played by primogeniture, if it has any effect in the production of malformations. If they show no tendency to occur more frequently in the first-born than they do in other members of the family, then we may justly question whether there is any basis for the belief that Nature makes more errors in her first attempts at reproduction than she does in her later ones.

The data on which the curves for the congenital cases are based were secured by collecting the first 1000 cases of congenital defects in the literature in which the place in family was stated. These

cases occurred in 987 families, because in thirteen instances, two widely different defects, such as meningocoele and malformed heart for example, occurred in two children in one family. On the other hand there were in these 987 families, 1008 first children, because in twenty-one instances, a normal and an abnormal child were born as twins at the first pregnancy. Hence it had to be recognized that there were two first children in those families, since both children had been subjected to the same intrauterine conditions, and to the same inexperience on the part of the uterus. Similarly, if the second or third pregnancy resulted in twins, one normal, the other abnormal, that family was credited with two second or third children. When the twins were stated to be uniovular, they were listed as one child, but if they were of opposite sexes, or of the same sex but obviously from two ova, they were listed as two children.

During the collection of the cases numerous references were found in which it was obvious that the malformed child was not the result of the first pregnancy, but for whom the place in family was not stated. For example, the report would read, "Delivered Mrs. S., a multipara, of an anencephalic fetus." These reports were at first discarded, and it was not until the first 300 cases had been found in which place in family was stated that a record of these "multiparous" cases was kept. These will be dealt with later on.

Mongolism was excluded from the list of 1000 cases for two reasons: first there is accumulating evidence to show that it is due to the inheritance of recessive factors, and second, it is usually stated that it occurs more frequently in the later pregnancies, owing either to reproductive exhaustion, or to too advanced age of the mother. Lest its inclusion obscure any tendency toward the more frequent exhibition of defects on the part of the first-born, cases of it were omitted.

The standard curve was plotted from the 987 families in which the 1000 defects occurred. There were 3748 members divided among fifteen places in the family. This curve is shown in figure 3. It will be noted that 27 per cent of these persons were first born in the family, a figure which is 6 per cent higher than that in the standard curve found in the cases of the hereditary defects. The reason is obvious. Many of the latter cases came from pedigrees extending through several generations, and in which most of the families were of course complete. Moreover many of the hereditary defects were those that did not appear until adult life, and so would not be apt to occur in the family when there was much possibility of more children

being added to its number. In figure 3, on the other hand, the defects are those noted at birth or shortly after, and thus they occur in a family where there is presumably still chance for subsequent children to be born.

When we plot the curve for the affected cases of this group, we find that 29.5 per cent of the affected were first-born. This then shows an increase of 2.5 over the percentage of first-born in the families

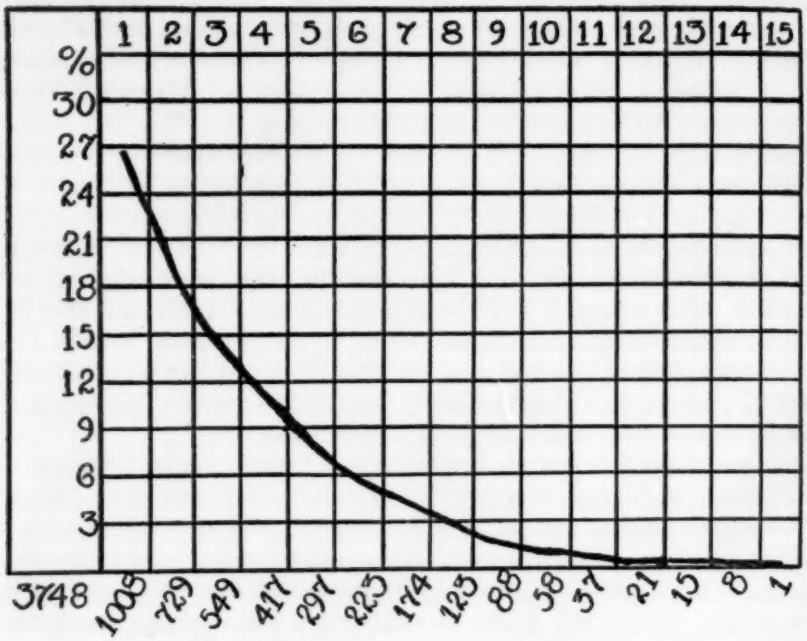

FIG. 3. CURVE SHOWING PERCENTAGES OCCURRING IN EACH PLACE IN FAMILY IN 987 FAMILIES COMPRISING 3748 MEMBERS, OF WHOM 1000 SHOWED CONGENITAL DEFECTS

as a whole. It must be remembered however, that there were found fifty cases of defects which were *not* the first but whose place in family was not stated. This means then that of 1050 cases of defective children, 295, or 28 per cent were first-born. It is this corrected figure for the percentage of affected first-born which is used on the curve in figure 4. When it is further recalled that all the "multiparous cases" found during the collection of the first 300 cases were discarded, and that the fifty used in the correction of this figure were found during the collection of the last 700, then the fully corrected

percentage in figure 4 for the first-born would be 27.5 per cent, on the assumption that a proportionate number of multiparous cases were found in the first 300 cases to those found in the last 700. But, after all, this further correction is not needed. Surely one per cent more cases of affected occurring in the place of the first-born than is warranted by the percentage of first-born in that population is not enough to call forth theories of uterine inexperience and Nature making mistakes in her first attempts at production.

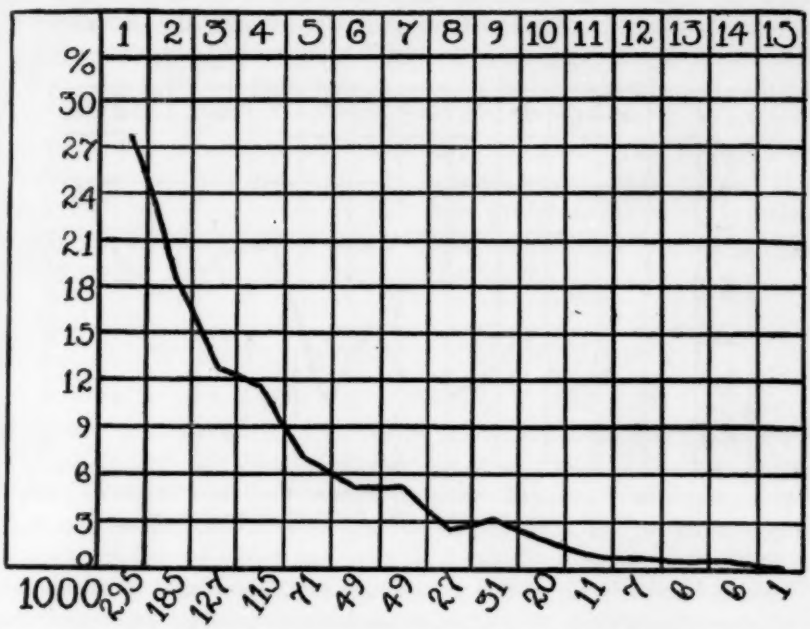

FIG. 4. CURVE SHOWING PERCENTAGES OF THE 1000 AFFECTED PERSONS OCCUPYING EACH PLACE IN THE FAMILY

It is true that, in figure 4, we have 8 per cent at least more affected persons falling in the place of first-born, than is warranted by Still's standard curve, or by the one plotted from a population of 1800 persons in figure 1. This might seem then to settle the argument in favor of the greater liability of the first-born to congenital malformations. Some at least of the families represented here are incomplete, and when more children are born, the percentage of the first-born in the population in the standard curve would drop still lower, while the percentage of affected in the first place in the family might remain the same,

thus increasing the discrepancy between the curves, and emphasizing still further the greater liability of the first-born to malformations. This may be overcome by plotting the curve as in figure 5. Here again we determine not what percentage of the 1058 cases occurred in the first-born of the family, but what percentage of the 1058 first children in the families considered were affected, and the same for the other places in the family. We find in this curve no increased tendency on the part of the first-born to develop congenital defects. The curve is a series of variations on either side a straight line, which deviations would become less the greater the number of cases collected.

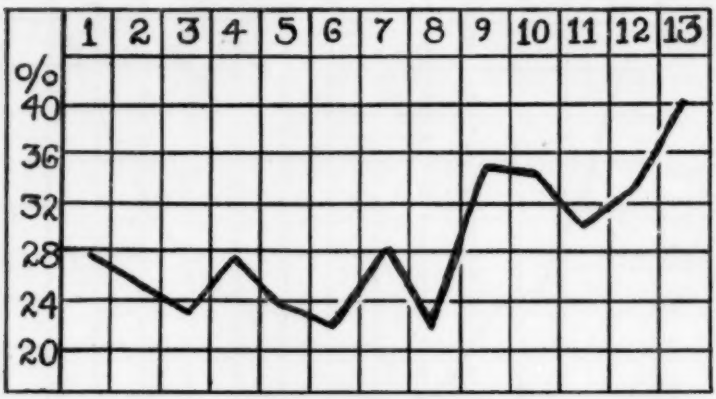

FIG. 5. CURVE SHOWING THE RATIO OF AFFECTED TO UNAFFECTED MEMBERS IN EACH PLACE IN FAMILY IN THE 987 FAMILIES LISTED ABOVE

There is evidently as great a percentage of affected among the fourth and seventh children of the family as among the first; and beginning with the ninth a constantly greater percentage of affected. Are we then to assume from this that the later children are more apt to be affected, perhaps due to reproductive exhaustion, or some other cause? No. It merely means that so few families have nine children or more that the figures are apt to be unreliable for so small a number of cases.

Of course, this curve might be criticised upon the basis that if more children were born in these families, and were unaffected, the value of every place other than the first would be lowered. This criticism is valid only when it is assumed that all the children yet to be born in these families will be normal, an assumption which is not justified, as will be shown later.

When we determine for figures 3 and 4 the average pregnancy as was done for figure 1, we find that the average pregnancy in these 987 families was 3.51 ± 0.027; and the average pregnancy which resulted in the malformed child was 3.58 ± 0.06. The difference between the averages is 0.07 ± 0.065. The probable error for the difference is almost identical with the difference itself, showing that these two curves are practically identical, and that the first child is not more apt to be malformed than any of the other children.

The data shown in figure 5 were used to determine the average ratio of affected to normal children in these families, and the deviation of the value for the first child from that average. It was found that the average ratio for these families was 27.91 per cent affected, ± 0.04. The value used in the computation of this average was the corrected figure for the percentage of first children affected, based on the 1058 cases, namely, 27.88 per cent. It is quite evident that this value is removed from the average by less than the probable error, in other words the first child in the family is affected no more frequently than is the average for all the children in the family.

CONGENITAL HYPERTROPHY OF THE PYLORUS

Especially with respect to this affection, Still holds that primogeniture is an aetiological factor in its production, and publishes a curve based on 400 cases to support his statement. This curve shows that 48 per cent of the cases of pyloric stenosis of congenital origin occurred in the first-born of the family, while it will be remembered that his standard curve had only 18.5 per cent of the population in the ranks of the first born. Among the 1000 cases which I collected there were fifty-nine cases of congenital hypertrophic pyloric stenosis. When the curve showing the percentage of affected in each place in the family was plotted for these cases, it was not very different from that plotted by Still from 400 cases, so that although there are some variations between the two curves, they are sufficiently similar to indicate that the fifty-nine cases which I collected were a fairly representative group. Figure 6 shows these two curves plotted together, the solid line being Still's and the broken line mine.

If now the curve be plotted so that it shows the ratio of affected to unaffected first-or second-born, as in the right hand side of figure 6, instead of the percentage of the affected which were first born as in the left hand side of the figure, we see in these fifty-nine cases at least,

that a smaller percentage of the first children in these families, in which pyloric stenosis occurred, developed this condition than did the second, third, fourth or fifth children. In other words, from this curve it would seem that if one were unfortunate enough to be born into a family in which hypertrophy of the pylorus was going to appear, one's chances were 65 per cent in favor of being normal if one were the first child, only 37 per cent if the second, 49 per cent if the third,

FIG. 6. LEFT SIDE OF FIGURE REPRESENTS IN THE SOLID LINE STILL'S CURVE OF AFFECTED OCCURRING IN EACH PLACE IN FAMILY BASED ON 400 CASES OF CONGENITAL HYPERTROPHIC PYLORIC STENOSIS WHILE THE BROKEN LINE IS BASED UPON 59 CASES COLLECTED BY THE AUTHOR. THE RIGHT SIDE OF THE FIGURE SHOWS THE RATIO OF AFFECTED TO UNAFFECTED IN THE 59 FAMILIES IN WHICH THESE 59 CASES OCCURRED

15 per cent if the fourth, and none at all if one were the fifth. Naturally these figures are not to be taken literally, or interpreted to mean even that there is an increasing tendency for the later members of the family to develop congenital hypertrophic pyloric stenosis. The most that I feel one is justified in drawing from them is this; that there is no greater tendency for the first-born of the family to develop hypertrophic pyloric stenosis than there is for any other child in the family to show it.

If this be true, how then are we to explain Still's 48.5 per cent and my 42 per cent of the affected being found among the first-born of

the family? The explanation is as follows. This affection is one which, because of its immediate disastrous results, is called to the attention of the parents and the physician in the first few weeks of the infant's life, and thus the case is brought to the physician's records while the family is still presumably incomplete. Thus in these families there is apt to be a far greater percentage of first-born children both normal or defective than would be met with in the population where the families were complete. If we examine the families of the fifty-nine affected cases here listed we find that there were 110 members, of whom fifty-six, or 51 per cent, were the eldest child. When we compare that with 42 per cent of the affected being first-born we see that there is exhibited a lessened and not a heightened liability on the part of the first-born to show this malformation. Here as elsewhere Still has made the mistake of comparing the curve of place-in-family of the affected persons with a standard one plotted from a population of which they were not a part, instead of with one made up of the families from which they came.

He explains this by saying that his "affected" curve would be altered very little, even if it were based on families which were completed, because that would bring the curve of the place-in-family of the entire group with which he was dealing nearer to the standard curve without disturbing the curve of place-in-family of the affected members, inasmuch as he found the condition of pyloric stenosis occurring but rarely in more than one member of the family, only 1–2 per cent of his cases showing familial tendency. Just what percentage of these cases occur in families in which more than one member was affected I cannot say, but in encountering the fifty-nine cases listed above in which only one member of the family showed the condition, records of sixteen other families were found in which a total of thirty-six cases were noted. Thus in seventy-five families, sixteen, or 21 per cent, showed familial tendencies; while if the *cases* be considered, of a total of ninety-five cases, thirty-six, or almost 38 per cent, showed other persons in the families afflicted. It seems unwise to say that the fact that the family is incomplete at the time that the record of it is made has little effect on the affected curve in the case of congenital hypertrophic pyloric stenosis.

With respect to this condition then, the evidence seems to warrant the following statement. There is no greater liability for the first child of the family to exhibit pyloric stenosis than there is for any other member of the family to show it. It is a condition that may recur in other members of the family when once it has shown itself.

CONGENITAL HEART DEFECTS

Of congenital heart defects, the series of 1000 cases contained but forty-five, these occurring in forty-five families consisting of 214 members. In figure 7 is shown Still's curve based upon 280 cases, drawn in the solid line, while mine based upon forty-five cases is made of dashes. There is wide disagreement here between the two curves, which would probably disappear to some extent were the number of cases in my series larger. Picked at random, however, as they were,

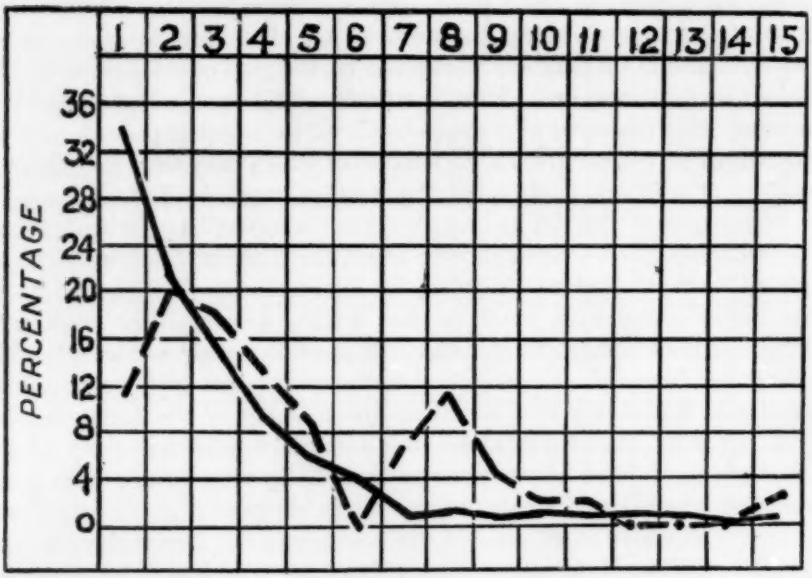

FIG. 7. SOLID LINE IS STILL'S CURVE SHOWING THE PERCENTAGE OF THE AFFECTED OCCURRING IN EACH PLACE IN FAMILY IN 280 FAMILIES SHOWING CONGENITAL HEART DISEASE. BROKEN LINE BASED UPON 45 CASES COLLECTED BY THE AUTHOR

they should show some tendency on the part of the first-born to be more frequently affected in proportion to their percentage in the population than is the case if Still's theory is correct. When the proportions of first-born and second-born which are affected are plotted as in figure 8, we find only one place showing a lower percentage of affected than the first, all the others showing an increased proportion of their numbers affected. I do not mean to state that the first-born is *less* liable to congenital heart defects than any of the other children in the family, but even from this small group of cases, unselected as

they were to prove or disprove any theory, we can question whether the first-born shows any *increased* liability to this affliction.

Owing to the relatively small number of these cases, objection might be raised that they were not representative, but inasmuch as the conclusions based upon 1000 cases are in agreement with those based upon this smaller number, we may be justified in assuming that these figures are not far from the truth.

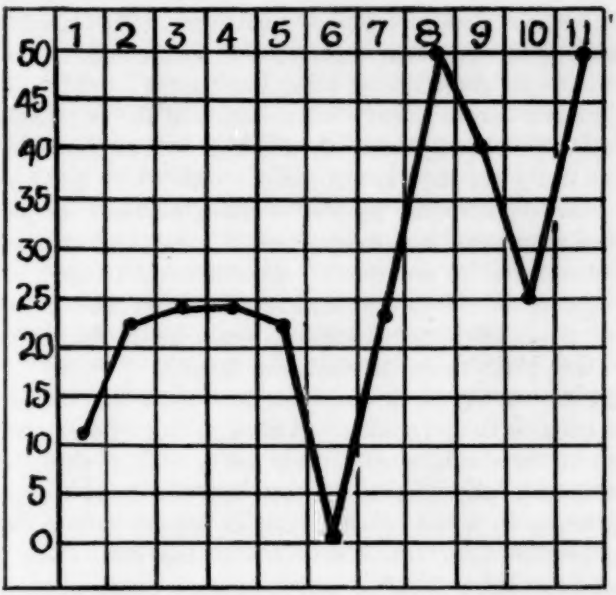

FIG. 8. CURVE SHOWING RATIO OF AFFECTED TO UNAFFECTED IN EACH PLACE IN FAMILY IN THE 45 CASES LISTED ABOVE

DISCUSSION OF RESULTS

Wherein do these results of mine based upon 600 cases of recognized hereditary disease, and upon 1000 cases of congenital defects, regarded by most writers as non-hereditary, differ from those of Still, who, from statistics gathered from his own practice, concludes that primogeniture is in itself a factor largely responsible for the appearance of malformations in the first-born and that it is highly improbable that subsequent children will show the defect? First of all he plots his standard curve from families to which his defective children do not belong, so that he is comparing his affected curve with one made up of a

population that is wholly apart from the families with which he deals. In the second place he feels justified in doing this because he assumes that no more malformed children will be born in the families which he is considering, stating that it is very exceptional for more than one malformed child to be born in a family. This assumption is far from valid, on the basis of evidence to be presented. He fails to recognize that frequently the reason for no more defective children appearing in the family, is that the parents have no more children, normal or defective, for fear of repeating their misfortune. He fails to make the curve which shows the ratio of affected to unaffected, in his families, and so misses this check on his other curves.

His standard curve shows 18.5 per cent of the population in the ranks of the first-born, while his affected curves show percentages well above thirty for the various defects with which he deals. Thus in congenital hypertrophic pyloric stenosis, he finds 48 per cent of the affected were first-born; in epilepsy, in mental deficiency exclusive of Mongolism, and in a series of miscellaneous congenital defects, 40 per cent were the eldest; and in congenital heart diseases, 34 per cent were first-born. These percentages indicate, he thinks, the part which primogeniture plays in the causation of disease.

His great error lay in assuming that no more malformed children would be born to these families. Although the following ratios may not represent the absolute truth, they are as reliable as similar figures would be from a physician's practice. In the hunt for 1000 cases occurring singly in a family, 167 cases of similar defects were found occurring in sixty-four families. This means then that for every fifteen families with a so-called non-hereditary malformation in them, there was one family, in which the same type of abnormality was familial in distribution. It means that for every six *cases* of these defects which occurred singly in a family, one case was found in which other members of the family were similarly afflicted. These percentages do not indicate that it is "very exceptional" for defects to be repeated in families, nor do they warrant our assuming that when one defective child has been born in a family, its troubles in that direction are over.

In the face of figures like that it is an error to compare the curve of affected persons with a standard curve in which the families are complete, assuming that no more affected children will be born to alter the affected curve.

MISCARRIAGES

Still discusses the proportion of miscarriages occurring in the first pregnancy, and finds that in this, as in congenital defects, there seems a greater liability on the part of the first pregnancy to end in miscarriage than do the later pregnancies, and that this is more marked in families where there is a single miscarriage. This finding is in direct opposition to that of Meyer and of Franz and Stumpf quoted by Meyer. The first two writers state that abortions are twice as frequent in women who have gone through former pregnancies as they are in primiparae. Franz found in a series of over 800 cases that less than five per cent of the miscarriages occurred in primiparae. This figure, in view of the fact that first births form an average of 20 per cent of all births, according to Still, indicates that the first pregnancy certainly shows no predilection towards early termination.

This supposed greater tendency to miscarriage during the first pregnancy is interpreted as an attempt on the part of Nature to check further progress in an unsuccessful experiment. That would mean that we should expect to find the greater proportion of miscarriages in the first pregnancy composed of abnormal specimens. In Meyer's series, which were examined not only for gross external abnormalities but for minute internal defects as well, there were 147 listed in which the families had no other children. This does not necessarily mean of course that the miscarriage occurred in the first pregnancy, but merely that there were no other living children. Of these only 46 per cent were abnormal, showing that for every abnormal pregnancy that is got rid of, there is a normal one terminated also.

Again one must be more than wary of basing conclusions upon the percentages of miscarriages occurring in *any* place in the family, for unfortunately one cannot say what percentage of these would have occurred had no attempts been made by artificial means, either mechanical or medicinal, to terminate the pregnancy. That many pregnancies are terminated early by abortion being induced has to be recognized; and because of the moral implications involved, such attempts are denied and the miscarriage attributed to falls or psychic influences or to some other fictitious cause. Hence conclusions based upon abortion statistics which interpret all miscarriages as efforts of Nature to get rid of mistakes, inevitably contain a large element of error.

"CONGENITAL" DEFECTS IN REALITY INHERITED

There seems then to be no more tendency for the first-born to develop congenital defects than there is for any of the other children. We can therefore question whether uterine inexperience can really be the cause of fetal abnormalities. Indeed when we remember that in 7 per cent of the first pregnancies which resulted in a deformed child, there was born a perfectly normal twin, we are unable to conceive of an inexperience which would affect one half the contents of the uterus while leaving unmolested the other half. We are forced to look to some other explanation than mistakes on the part of the uterus, and the natural place to look for the abnormality is in the ovum itself. There is a great deal of evidence to support the theory that these so-called "congenital" defects are in reality inherited. Still comes to this conclusion for some of the cases with which he dealt, saying that when pregnancy after pregnancy resulted in children with the same malformation, some other factor more far reaching than primogeniture must be at work. The probabilities are that even when only one child in the family is affected, the same underlying cause, heredity, is responsible. The following facts lend strong support to the theory that all malformations are hereditary. When the twins appear to have been derived from one ovum, and are of the same size, any defect in one is duplicated in the other. If the twins are derived from two ova, defects in one are not necessarily found in the other, although both were subjected to the same intrauterine environment. The same defects may appear in children of the same family not born at the same time; Ballantyne has stated with respect to anencephaly that a woman who has produced one such malformed fetus is apt to produce more of the same kind. Nor does this statement apply alone to anencephaly. As stated above, for every six cases of malformation occurring singly in a family, there was one which was characterized by its familial distribution.

One may ask if *all* such cases are hereditary, simply because some appear to be so? If inherited why do not a greater proportion show familial distribution? The answer to the latter is probably this. The defects are dependent upon a number of factors, probably recessive in character, and not until a combination of all these is made does the malformation appear. Thus the chances are that in small families the proper combination is not made oftener than once, if at all. As to the former question, it seems quite probable that local conditions

might be the cause of some of these abnormalities; but the great number of records which state that the individual was in perfect health before and during the pregnancy would indicate that local uterine troubles are not at the basis of all the malformations.

One of the strongest arguments in favor of the hereditary nature of these defects is the following. As is well known the tendency to twinning runs in families, being transmitted through both male and female lines. Now Stockard has shown that in the fish egg (and the

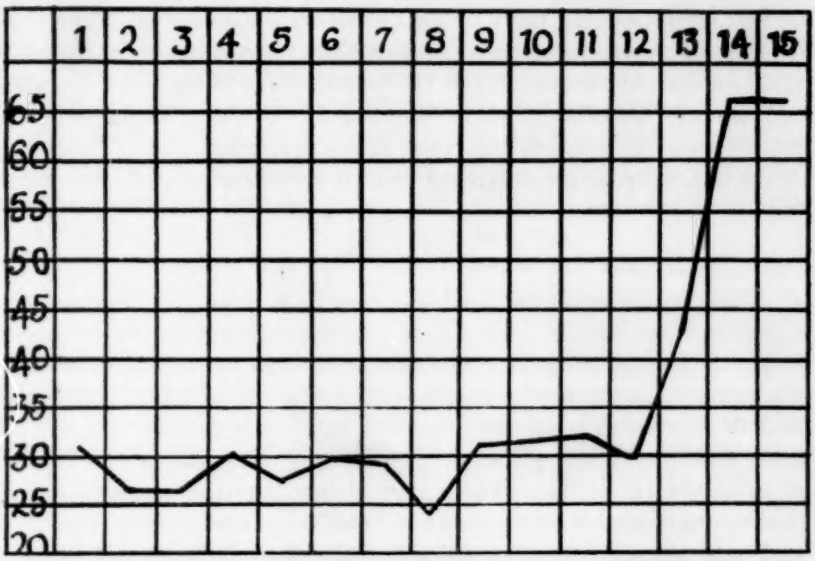

FIG. 9. CURVE BASED UPON THE COMBINED CONGENITAL AND HEREDITARY CASES LISTED ABOVE, SHOWING THE RATIO OF AFFECTED TO UNAFFECTED IN EACH PLACE IN THE FAMILY

arguments are applied to mammalian forms as well) the same type of stimulus which produces normal uniovular twins, is responsible for the formation of monstrosities, which either lack or have duplicated certain portions of the body. If these two statements be true, may it not be that the tendency toward the production of monstrosities is inherited as is the tendency to multiple pregnancies? The site of the inheritance is not in the uterus which fails to respond normally to the imbedded ovum, but in the ovum itself, since the inheritance of the defective structure may be transmitted by the father as well as by the mother.

Thus, although I have listed many defects under the heading "congenital" and discussed them apart from those that are well recognized as being hereditary, this was done to conform to the opinion of most writers that these developmental anomalies are not inherited, and to see if there was any difference between them and the inherited group of defects as far as the frequency with which they affected the first-born is concerned. A glance at figures 2 and 5 shows that the curves illustrating the percentage of children which are affected with these two sets of malformations are very similar, thus further strengthening the idea that they are all inherited. The data for the two groups have been fused and the curve plotted for the ratio of affected to unaffected in the 1600 cases of hereditary and so-called "congenital" defects. This is shown in figure 9, and the curve represents a series of deviations on either side of a straight line. The values in the last three places are based on so few cases as to make the curve unreliable here.

SUMMARY

There is no evidence derived from 1000 cases of "congenital" malformations usually regarded as non-hereditary that the first-born is any more apt to suffer from developmental defects than are the the other children in the family. The laws of heredity presuppose that the first-born is no more liable to hereditary defects than are the other children, a theory that is supported by the evidence of 600 cases of inherited anomalies. There is evidence that the congenital malformations regarded by most as non-hereditary, are in reality inherited and to be classed with other inherited defects. They are apt to occur in more than one child in the family. Hence advice given to parents, that primogeniture is in itself a large factor in the production of malformations, and that it is "highly improbable" that any of their subsequent children will be defective if they have been unfortunate enough to have their first offspring malformed, is pernicious, in that it raises hopes that may be tragically disappointed, and because it is not warranted by the observed experience of many physicians.

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REVERSION TO PRIMITIVE BEHAVIOR RESULTING FROM ORGANIC DISEASE OF THE BRAIN

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It has long been recognized that focal lesions of the brain are associated with disturbances in behavior, but while the phenomena of such disturbances have been abundantly described, their interpretation has lagged behind. The synkinetic movements in hemiplegia, for instance, though listed in great detail, have only in the last few years been recognized as postural reactions. The opisthotonos of acute meningitis has been featured in the clinical descriptions of the disease, but its relationship to decerebrate rigidity was only recently pointed out by Wechsler. The purposive adaptation of the flexion reflex of the paralyzed lower extremity was indicated by Babinski in his well known paper on reflexes of defense. I shall attempt in this paper to suggest the nature of some of the automatic responses observed in cases of disease of the brain, and to show how the behavior of the individual with a focal lesion may resemble the behavior of the normal individual during earlier stages of development.

These reactions have certain features in common. They are involuntary, the latent period is short, the response is vigorous and quite stereotyped, often rhythmic. Some are best provoked by *painful* stimuli, deep scratching, compression, over-extension of joints. Others depend upon great bodily exertion by the patient. They seem to be adapted to self-preservation, yet they are imperfectly differentiated for the preservation of the man in his adult state of development. Going back to more primitive beings, human and subhuman, however, one can see that they might serve satisfactorily as protective mechanisms.

These reactions are not elicitable in every case of organic cerebral disease. The powers of compensation on the part of the brain are so

great that an injury must be quite extensive for permanent effects to appear. Moreover, paralysis is often followed by contracture that prevents movement. On the other hand the human cerebral cortex is so predominantly an executive organ that injury to it has much greater consequences than a similar injury to the cerebral cortex of a lower animal like the dog. Generally speaking the reactions are more vivid when the injury takes place before complete development of the nervous system. Postural reactions are a necessary phase during development, but as the cortex becomes dominant it suppresses many of these postural mechanisms in the interest of economy of effort and greatly extends the range of individual movements in the segments of the limbs. The transfer of authority from lower to higher centers is associated with regression in the capabilities of the lower centers to carry on when they are left in control through disease of the higher ones. Therefore the individual who has successfully passed through the stage of postural mechanisms and developed economical cortical control will be less likely to develop any considerable postural reactions when the cortex is damaged, than would one whose cortical control never developed to any great degree.

THE BEGINNINGS OF MOVEMENT

In order to recognize the primitive character of these reactions it will be well to trace the development of bodily movements from their initiation to their stage of perfection. This has been followed best and best correlated with the development of the primitive nervous system by Coghill in the amblystoma embryo. A short description of the behavior of this salamander will throw light upon the development of behavior in the vertebrate series. The newly hatched embryo consists of head, body and tail with no sign of limbs. Its only reaction is to twist itself into a single coil due to simultaneous contraction of all the muscle segments on one side of the body. The reaction is simple, complete, stereotyped, and may be elicited time after time by a suitable stimulus. It serves in its primitive way to save the animal from harm but its lack of purposive adaptation is shown only too well in the failure of the animal to escape from the irritant. A little later this simple coil reaction is replaced by the double coil, a twist to one side being succeeded immediately by a twist to the other side, turning the embryo into an *S*-shape. Rapidly succeeding, alternating coil reactions will convert the static individual into a free-swimming one,

thereby enabling the embryo to escape from the neighborhood of the exciting stimulus. Not long after the embryo passes into the free-swimming stage, limb-buds make their appearance, moving with the body, but as yet possessing no individual control. Even without separate movement, however, these limb-buds are of advantage to the animal, since they serve to catch upon projections. If the animal is placed upon a dry rough surface before the limbs develop, although it goes through the same swimming movements that it makes in the water it merely wriggles like a fish and does not progress. When the limb-buds develop, a coil in one direction will bring the fore and hind limbs into close apposition on the concave side while it separates the opposite limbs. An alternation of such twistings, with the leverage allowed by the extending limbs rather rapidly gives the animal power to move about on a dry surface even without control of the individual segments. The power to move on land gives the animal additional means of escape, and is better adapted as a response to nocuous stimuli than simple coiling or even free swimming. With the development of the nervous system, control is gradually gained over the individual limbs so that the adult salamander not only has no need for remaining in the water, but is able to lift itself off the ground and run in a rather clumsy fashion. The feet still open and close as units, but the alternate spreading and bringing together of the digits serves well in swimming, since it supplies a larger area of resistance on the stroke and a smaller one on the recovery, and on land it increases the stability. There still remain many reactions to be acquired, notably the control of the separate segments of the limbs, but to observe these we must go higher in the scale of animal life.

From amblystoma to man may seem a long jump, yet I regard this preliminary consideration of the lower animal as a necessary step in the understanding of motor behavior. We are not well acquainted with the character of the primordial movements of the human fetus, although from the fifth month on they are readily perceived by the expectant mother. The youngest moving fetuses recovered have shown convulsive movements somewhat analogous to the simple coil reactions of amblystoma. It must be remembered, though, that the amphibian can be examined in its own element, whereas the fetus is at the point of death, being unable to live separated from the mother for more than a short time. Fetuses of older age show a much more varied response to stimulation, so that at birth the infant is endowed with a rather complex form of behavior. Cur-

iously enough the reactions of the new-born infant have received rather limited attention. Here is a vast field for painstaking physiologic and psychologic research.

A few elements in this behavior are worthy of note. Certain of these are destined for survival as long as the individual shall live; other elements will disappear in the succeeding months never to reappear; but a large number of reactions will disappear within weeks or months, and then, should the individual suffer some injury to the brain, will reappear when the effective stimulus is applied. The stable reactions that are present at birth and will normally remain to the end are such actions as breathing, swallowing, winking, urination and defecation, vocalization. Actions that disappear permanently are relatively few, and their number is decreasing as clinical research progresses. The rapid rhythmic sucking on anything pleasant placed in the mouth, the spasmodic throwing out of the arms when the child is allowed to fall backwards, the generalized doubling up and tremor during crying—such reactions seem to disappear as the child develops, never to return again. In the intermediate category of reactions normally outgrown but reappearing as a result of disease of the brain there are many defensive reactions, postural reactions and even more highly integrated responses in the emotional and intellectual spheres.

THE FLEXION REFLEX

The best known of these reactions is the dorsi-flexion of the great toe and withdrawal of the lower limb when the sole of the foot is scratched. This reaction is very primitive and yet evidently highly useful to the animal economy, since it is preserved intact by all the suprasegmental mechanisms up to the motor cortex itself. With the development of the motor cortex the movement of the great toe is converted from dorsal extension into flexion, and so persists normally to the end of life. Anything that interrupts impulses from the paracentral lobule to the lower lumbar segments of the spinal cord will cause the reappearance of this primitive defensive mechanism. This reaction is by no means a simple reflex. The spread of activity to different muscle groups as well as the wide reflexogenic zone, is indicative of a complex reaction. The flexion reflex is most active in cases of complete destruction of the spinal cord in the thoracic region. Following a noxious stimulus to the sole of the foot there is dorsal extension of the great toe, fanning of the other toes, and brusque

flexion of the lower limb at hip, knee and ankle, drawing the foot many inches from the point of application of the stimulus. There is sometimes an extensor thrust of the opposite limb. In exceptional instances of great activity in the autonomous spinal cord there may even be reflex evacuation of the bladder and rectum, with an outbreak of sweating. Moreover, the stimulus causing this reaction may be effective when applied not only to the foot but also to the leg, the thigh, and even to the abdomen, provided it is below the level of injury. Filling the bladder with water through a catheter will occasionally provoke spasmodic flexion of the lower limbs. This total reaction or "mass reflex," as it was called by Head and Riddoch, can be provoked in limbs that are completely powerless and completely anesthetic as far as the patient is concerned. Somewhat similar reflex phenomena have been described in the upper limbs when the injury to the spinal cord has been located in the upper cervical region. Observations are few, however, because of the greater mortality following injuries in this region.

The flexion reflex is well known in infants. The extensor thrust has not been investigated. I have observed emptying of the infant's bladder follow scratching the sole of the foot. It is interesting to speculate upon the use of such a mechanism in the conditioning of young children. Probably the reflex nature of the reaction disappears too early to be of practical use, but if not it would ease the task of many a mother.

The flexion reflex is preserved until the motor cortex enters into operation. This means that it is embodied in a number of more complicated activities presided over by such suprasegmental mechanisms as the cerebellum, the red nucleus, the reticular formation, and possibly even the basal ganglia. This reaction, the so-called Babinski reflex, forms an integral part of more complicated types of behavior that are perhaps well adapted to the preservation of the individual under certain conditions, but are nevertheless inadequate when compared with those possessed by the normal adult.

POSTURAL REACTIONS

In taking up the simpler postural reactions that appear as the result of cerebral disease we enter a field whose normal prototype in the developing child is largely untilled. Much more is known about these reactions from the study of diseased adults and from experi-

ments upon animals. It seems necessary for their appearance that the medulla oblongata and the pons be intact. The medulla is a terminus for many fibers from the spinal cord, and others from the vestibular nerves. The impulses conveyed by these fibers are integrated in the medulla and find expression through different pathways originating in Deiters' nuclei and the reticular formation. As long as inhibitory impulses are allowed to come from higher centers such as the red nuclei, the distribution of tonus is fairly normal. When these are cut off, however, tonic reflexes are set up which depend for their pattern upon the position of the head in space (vestibular reflexes), and in relation to the body (cervical reflexes). As a rule, following transection of the nervous axis below the level of the colliculi, the limbs of the experimental animal become rigid, reflexes are increased and the postural pattern is rigidly determined by the position of the head. When the head of such a rigid animal is rotated from one side to the other, the limbs on the side toward which the occiput is turned become flexed while the opposite limbs undergo extension. When the head is bent forward the limbs all become flexed, when it is bent backward the limbs are all extended. Such reactions disappear if the medulla is injured, or if afferent stimuli are cut off by sectioning the cervical and vestibular nerves. Equivalent behavior may be seen in patients who have temporary or permanent interruption of all tracts leading from the brain to the centers of the pons and medulla. Thus in the tonic phase of the epileptic attack there is a period of rigidity of all four limbs in which the posture may be varied much in the manner of the decerebrate animal, by rotation of the head. Also in certain patients with tumors or hemorrhages at the base of the brain such a condition of "decerebrate" rigidity may be encountered. The opisthotonos of meningitis is an exact counterpart. Can we recognize such states in the normal infant? Probably the development of the nervous system is so delicately adjusted that "decerebrate" rigidity never normally occurs, yet to the observer who knows what to look for, it is not difficult to perceive minor signs of the major condition. For instance, the emotions and strong muscular activity have a way, as it were, of short-circuiting the nervous system and permitting the appearance of fundamental mechanisms that are normally overlain by complex and better adapted defense reactions. More concerning the emotions will be developed later. For present purposes it is sufficient to call attention to the behavior of the squalling infant. The succession of doubling-up and concerted thrusts will

resemble more or less closely the generalized tonic contractions of all the limbs in the animals and sick individuals noted above. The states of general extension are too evanescent to allow examination of the cervical reflexes, but the labyrinthine reflexes show up quite well. They do this even in the normal infant who is not crying. Hold the baby face down supported by the trunk, with the head and limbs free. Provided the child has reached the stage of holding up its head—which many infants can do at birth—the neck, back and limbs will all be quite strongly extended. If now the head is pushed foward, the baby folds up like a clasp-knife, with flexion of all the extremities. Extending the neck again provokes extension of all the limbs.

These postural reactions, at least in the typical cases, are of tonic character and tend to persist as long as the abnormal position of the head is imposed upon the subject. Ordinarily such primitive postural reactions are not seen, since they are overlain or altered by reactions of a higher order, or are of only momentary duration. There are certain postural reactions, however, that appear in the limb devoid of cortical control, and probably depend upon the functional integrity of the great tone centers in the midbrain, hence Morin and I have termed them reflexes of mesencephalic automatism.

SYNKINESES OR AUTOMATIC ASSOCIATED MOVEMENTS

The patient with hemiplegia holds his paralyzed arm in a peculiar manner and in walking moves it involuntarily and more or less rhythmically but not in the manner of the healthy arm. With each step the arm is brought away from the side, flexed at the elbow, and somewhat supinated, with the hand closed and the thumb opposed to the fingers. Provided no permanent contracture of muscles has taken place there is a rather marked tendency for this arm to move involuntarily in response to such general muscular actions as yawning, coughing, sneezing. Sometimes the responses vary particularly if the type of activity is altered. A paroxysmal action like a sneeze is apt to be associated with a sharp generalized contraction of the musculature on the paralyzed side bringing the arm close to the body with the hand clenched. A yawn on the other hand sometimes causes stretching out of the hand, which is under little or no voluntary control, together with spreading of the fingers in much the same way that the claws of a cat are spread during yawning. These acts are purely involuntary and often surprise the patients greatly.

The paralyzed arm may be galvanized into action by muscular exertion on the healthy side. This requires not only that the patient shall retain good mobility of the limb but also that he shall put a lot of effort into the action. It is this putting of all force into the movement that brings into play the postural centers operating upon both sides of the body. The play of such postural reactions is superbly demonstrated in track and field sports, where the "form" that is so much in demand is the proper postural reaction for getting the most out of the muscles used. Every muscle in the body, even in the face, is brought into the effort of putting the shot. To elicit these reactions in cases of cerebral disease, therefore, the patient must put a lot of energy into the voluntary action. The reactions are more easily elicited in children, whose postural reactions are more widespread than those of the adult. As I explained earlier, these reactions are total reactions, as contrasted with the reactions of individual control that are built up through practice and individual differentiation.

If a patient with ordinary hemiplegia is asked to pull hard against resistance, the paralyzed arm and leg will be activated by certain involuntary movements that are termed synkineses. As a rule there is increased flexion of the elbow with elevation to the shoulder level, and closure of the hand. The lower limb on this side becomes flexed at the hip, knee and ankle. If the patient is directed to push hard there is a tendency for the paralyzed arm to be thrust in extension across the body with the fingers closed into a tight fist. Here are two opposite postural reactions of retraction and protrusion in the paralyzed limb, brought out by activities of similar force but of opposite sign, namely pulling and pushing. In some cases the sign of the postural reaction remains uninfluenced by a change from pulling to pushing. The sign in such cases may be changed by turning the head, in other words by reinforcing the synkinetic reaction by means of the tonic reflexes of the neck. Rotating the chin toward the active hand will send the paralyzed arm high up in flexion, while rotating it in the opposite direction will cause the paralyzed limb to be thrust across the body in extension. This indicates that the tonus in the muscles of the body, especially in those of the neck and shoulder girdle, determines the postural pattern in the various segments of limbs that are no longer under differentiated control. Moreover, the nature of these automatic movements of opposite sign becomes manifest if the patient is placed prone supported by the trunk. Alternate pulling and pushing with the healthy limb will, through postural

reactions activated and determined by the musculature of the trunk, impose upon the paralyzed limb global movements of protrusion and retraction of the same sign as those in the active limb. That these movements are not acquisitive in character seems clear since the outstretched hand is firmly closed, and when the arm is retracted it is carried away from the body at the shoulder. I believe that in themselves these movements in the paralyzed limb represent those of weight bearing and quadrupedal locomotion, although the fact that the movements are of the same sign as those of the active limb shows what great involution this particular functional activity has undergone. Our paralyzed patients cannot walk on all fours, although on hands and knees they get along quite well. An instance such as this shows that with the suppression of individual control of muscle groups of the limb due to cortical injury there reappears an ancient mechanism going back even farther than the primate family, a mechanism that is the underlying coordinated act making locomotion possible. There are two additional points to be noted in this connection, first that running on all fours is not infrequently noted in children at about the age of one year—before walking upright becomes possible—and secondly that the mechanism is rarely complete in man although perfect in most lower animals following destruction of the cortex. The mechanism underlying running on all fours is evidently controlled by centers lying below the cerebral cortex.

Turning now to minor evidence of the postural nature of the reactions observed in the paralyzed limbs we note several signs of lack of individual control over the muscles of the limbs, but the presence of well marked synergic actions. The patient cannot raise the weakened lower limb from the bed without flexing it at the knee and ankle. When he stoops forward there is involuntary bending at the knee. Extending the wrist causes flexion of the fingers. When he reaches across his body the hand closes in a fist. When he stretches his arm above his head there is a tendency to spreading of the fingers and pronation of the hand. This last named postural position would seem to be adapted to arboreal existence. The pronation of the forearm is particularly impressive, since the apes grasp branches from the near side instead of from the far side as man does. Fine and rapid movements of individual fingers are difficult. We may find a similar type of behavior in the child that is attempting to sew, to draw, or to play with blocks. Movements are slow, jerky, inaccurate. The hand opens and closes as a unit. Only later does the child gain

control over the movement of separate fingers, and even in the average adult this control is far from perfect when compared with that of the accomplished pianist.

Thus far I have considered postural reactions presided over by lower levels of the central nervous system, reactions which normally disappear in the adult human, but which may reappear as a result of damage to the cortico-spinal system of neurones. This is obviously not the only higher system of voluntary motor control, indeed it would seem to be the most primitive of several higher systems. That the cortico-spinal system is most important in the control of voluntary movement is generally admitted, but defects in other closely related systems exert a damaging effect upon the freedom of voluntary motion presided over by the cortico-spinal system. In some respects the cortico-spinal system is only one important final common path for other superior mechanisms. The whole frontal lobe is an important center for complex motor behavior. Disease of the frontal lobe causes a very striking reversion to primitive behavior.

REFLEX GRASPING AND GROPING

A symptom which has interested me greatly during the past year is that of involuntary or reflex grasping, a phenomenon which may be seen best at the two ends of life. When the finger is placed in the infant's palm it is grasped, and the grasp tightens if the attempt is made to withdraw the finger. This is a very primitive reaction found wherever prehensile digits are present. It enables the young to hold to the mother during her travels, and the instantaneous tightening of the grasp when there is a tendency to slip is obviously a defense reaction against losing hold during a sudden jolt. This phenomenon persists for a number of months after birth as anybody who has watched children at play will be able to testify. In attempting to place blocks in a row or on top of one another the child often fails on account of the invincible tendency to hold onto the block in spite of conscious efforts to let go. In somewhat younger children an object placed in the hand will be retained until some other object claims the attention. Any effort to withdraw it meets with an increase in the force of the grasp which is not due to a desire to retain the object but is a reflex action over which the child has no control. Study of the facial expression of the child when the mother is trying to get the teething-ring or the cap-strings from its hand will show that the baby

attaches little value to the object thus held. The reaction wanes during the first year of life, but traces of it will remain until the close of the second year.

The phenomenon of reflex grasping will reappear as a result of disease of a certain part of the frontal lobe. In such patients an object brought into the palm is grasped, and the grasp tightens when the attempt is made to withdraw the object. The reaction is quite stereotyped, will be repeated again and again as the stimulus is repeated, the latent period is short, the fatigability is slight. The grasping is coordinated, the thumb being opposed to the fingers, and the force may be sufficient to crush fragile objects. Such patients if sufficiently intelligent will bewail their inability to let go. The reaction may be inhibited by applying a painful stimulus, such as giving the patient a cork stuck full of pins, or by directing him to do something that will distract his attention from the clutching hand.

The syndrome is most pronounced in cases of bilateral atrophy of the frontal lobes. I have studied a number of these cases and have verified the lesions at necropsy. The patients are usually aged and greatly demented, often unable to speak intelligibly. They lie placidly in bed unconscious of passing events, often apparently asleep, at other times gazing vacantly ahead of them. Any object that grazes the palm is grasped, and the grasp tightens if the object is withdrawn. When the hand is stroked on the back or edge it gropes for the object and by successive stimuli may be led back and forth across the bed. The nurses have difficulty in caring for such patients on account of their tendency to grasp the bedclothes, the chemise, the hand of the nurse. Also in the feet a tendency for the toes to fold over the stimulating object is somewhat the same as that observed in the infant. The feeding behavior of such patients is very interesting and is almost the exact counterpart of that of the infant four to six months of age. No matter what the type of food, any large morsel is immediately rejected by the combined action of the tongue and lips, although small morsels are taken with avidity. During feeding the hand may grope about catching the utensil or the arm of the nurse. Instead of carrying the morsel to the mouth, the patient pulls it away. I tried one patient with a nursing bottle. She evidently enjoyed it, taking about four ounces of milk. During the trial she groped about with both hands, caught the bottle, but instead of holding it in the proper way pulled it away from the mouth although clamping the nipple with her tooth-

less jaws until there was imminent danger of inundation. Rhythmic sucking movements were not observed, but it is possible that a larger nipple would have evoked these.

This set of phenomena requires the integrity of the cortico-spinal system of neurones. It is not present in ordinary hemiplegia, and if the disease of the frontal lobes extends to the central convolutions, the reaction disappears. There is abundant evidence that the frontal lobe has to do with movement, its connections with the motor system may easily be traced through the pons and cerebellum, but its function would seem to be a higher integrative one permitting the proper handling of objects. When the mechanism is interfered with there is reversion to a primitive type of reaction characterized by reflex grasping and groping with slowness, ataxia and concomitant phenomena.

PATHOLOGIC LAUGHING AND CRYING

Earlier in the paper I hinted at the effect of the emotions on muscular activity. Are there primitive emotional reactions that reappear as the result of disease of the brain? The answer must be in the affirmative, although in this case the localization of the diseased areas is less exact, since in all cases examined so far a multitude of lesions has been found. Not infrequently in these cases there are signs of damage to both cortico-spinal tracts, together with interference with the power of articulation, sphincter control, etc. Such patients are usually aged, with arteriosclerosis of the brain, and frequently their mental processes show loss in acuity. In them we observe uncontrollable outbursts of laughing or weeping, or a mixture of the two, continuing for a prolonged period even though the patient does not experience the emotions he is expressing. Any attempt at speaking or at performing some other rather complicated task will send such a patient off into gales of laughter or paroxyms of sobbing so that he is utterly unable to perform the act. Even control of the sphincters may be lost. Spasmodic laughing and crying are, in a way, primitive motor reactions which are brought under control early in life, but under certain conditions, disease of the brain permits their reappearance through suppression of the higher controlling centers, and then the whole mechanism may be touched off by a very slight stimulus. Their resemblance to the laughing and crying in the infant is perhaps not absolute, since the emotions of the child are probably real although the control is slight. However, the appearance of such phenomena

in the adult individual as the result of organic disease of the brain should arouse the psychologist to the possibility that the expression of the emotions in young children may be disproportionate to the real feeling tone present. As far as the relaxation of the sphincters during extreme hilarity is concerned, we may find a detailed account told with gusto by the noted Rabelais. The emptying of the bladder during laughing and crying in young children is frequently noted.

PRIMITIVE SENSIBILITY

I have dealt thus far with the reactions on the motor side, showing their dedifferentiation and return to a more primitive type of response following damage to the cerebral mechanisms. There is also some evidence on the sensory side of a return to a more primitive type of perception following disturbance of the cortex. A patient who has sensory disturbances following a stroke may be able to tell from handling an object whether it is warm or cold, hard or soft, pleasant or unpleasant to handle, but he will be able to gain no clear idea of the shape, weight or size of the object, nor will he recognize its consistency, texture, or roughness—perceptions upon which we depend for the recognition of unseen objects. We are dependent largely upon our imaginations for the recognition of an analogous phase in the development of the human individual, since the development of perceptions and of the use of symbols such as words is so intimately associated. We can, however, reproduce for ourselves some of the difficulties that such patients experience in recognizing objects with their paralyzed hands by the use of unfamiliar objects. There is a Hallowe'en game in which people sit in a circle on the floor with a large blanket covering their laps, and in which strange objects are passed from hand to hand. An apple stuck full of toothpicks, a rubber glove filled with sand, even a hot water bottle, become fearful and unknown objects highly charged with feeling tone. It must be admitted, however, that the surroundings are not particularly conducive to calm analysis of the attributes of the individual objects. Damage to the sensory cortex suppresses powers of discrimination, leaving a primitive type of sensibility in which pleasantness and unpleasantness are major criteria.

APHASIA

In the process of symbolic formulation and expression, in other words, language, we may also witness a reversion to primitive behavior following injury to the brain. Henry Head pointed this out in his

studies on wounded soldiers. There is a lapse in formulation from the abstract to the concrete. Common objects are always more easily recognized and named than are colors. Colors are qualities and therefore abstractions, so that in certain of his cases the abstract was replaced by the concrete in such terms:

Green—what grass is like	Red—like fire.
Blue—the sky	Black—what you do for the dead.

In a like manner, many patients are unable to repeat correctly the observer's actions when he is seated in front of them, but if the observer sits behind the patient and allows his movements to be seen in a glass, the patient seldom fails to copy the movements exactly. Direct imitation requires no construction of a mental picture of the act to be performed, and since it is the mental picture, or the symbolic formulation, that is disturbed by the disorder of the brain, the elimination of this step facilitates the correct completion of an ordinarily simple task. The child's expression of the abstract in terms of the concrete is a common phenomenon. Disease of the brain interferes with symbolic formulation and expression to the extent that symbols become tools difficult to use, yet when symbols are replaced by objects little difficulty is noted. Here again, however, so many objects are endowed with symbolic importance that generalizations must be closely watched lest they lead astray. Consider the symbolic importance of money, of books, of photographs. Books, for instance, are books to the bibliophile, to the librarian, to the trashman, and to the aphasic. To the author and to the reader, a book is a highly symbolic object strongly charged with intellectual and emotional tone. It transcends its objective value and becomes symbolic. In order to appreciate its symbolic value, however, that part of the cerebral cortex presiding over symbolic formulation and expression must retain its integrity.

DIFFERENTIATION AND DEDIFFERENTIATION AS RELATED TO BEHAVIOR AND THE NERVOUS SYSTEM

Throughout this paper I have attempted to show that behavior, including perception and symbolic formulation, is the outcome of differentiation in neuromuscular equipment, and cortical control. To take a gross example, limbs are not engrafted as units, first functioning as independent mechanisms and later brought into harmonious coordination with the rest of the body. They develop with the body,

moving at first only with the body in the same fashion that our ears move with our heads. Later they exhibit global movements of retraction and protrusion, movements of fixed pattern dependent for their sign upon the relative tone of the dorsal and ventral axial musculature. Progress is made through various stages of separate control over the different segments of the limbs, until the most distal units, the digits, are individualized and obey the directions of the master-organ, the cerebral cortex. Such control is not gained in a year or two. The act of rising to the feet from the supine posture in the adult manner is not acquired until the seventh year; writing, riding a bicycle, playing a musical instrument, are even later acquisitions. Even slower in development is intellectual behavior.

When cerebral disease disorganizes the centers controlling the more complex highly individualized components of our behavior there is still a substratum of postural reactions to fall back upon, simpler coordinations controlled at inferior levels, and means of gaining information about objects in the environment depending for their effect more upon emotional consciousness than upon intellectual appreciations.

The effect, then, of focal destruction of some part of the cerebral hemispheres is to cause a regression, a dedifferentiation of function to a lower order, but a function for which a counterpart may be observed in the individual whose nervous system is as yet immaturely developed. Such a concept brings the onus of responsibility upon those who are interested in examining the immature individual. Perhaps it would be better to say that a closer cooperation between those examining the developing individual and those examining the decadent individual would be more fruitful of results than the independent labors of either group alone. The subject of evolution and dissolution of the nervous system was begun on broad lines by Hughlings Jackson. We have come a long way from that period, have made certain advances, but there is still a rich mine of hidden treasure, requiring merely a little curiosity and a boundless supply of patience to yield luscious fruits of research.

NOTES

NOTES ON MONGOLIAN CRANIOLOGY*

In the American Journal of Physical Anthropology, 11, page 280 *et seq.* Mr. John Cameron follows up his previous craniometric studies with an examination of the Mongolian crania in the U. S. National Museum. He there gives the various average dimensions with their standard deviations, etc.; coefficients of correlation of some of the dimensions to the cranial capacities are also given. The conclusion is reached that the material is quite uniform, that from east to west, starting with Greenland, dolichocephaly gives way gradually to brachycephaly. The computations in this paper were made some time before the appearance of Mr. Cameron's article. When this appeared I was first inclined not to publish them, but a study of Mr. Cameron's paper has prompted me to present the data with some comments. That dolichocephaly gives way to brachycephaly from east to west may be granted; the uniformity of Mongolian and Greenland crania also may be accepted. But, for reasons given below, Mr. Cameron's figures and mine do not agree. The picture of the transition is decidedly altered. The figures suggest that the coefficients of correlation of skull-length to cranial capacity, etc., might vary from those given by Mr. Cameron.

The following table has been computed from the data contained in the Catalogue of Human Crania in the United States National Museum Collections, The Eskimo, Alaska and related Indians, North Eastern Asiatics (1924). The figures have been carried out further than is usual, for certain unexpected results have turned up.

The only correlations that are definitely significant are those of skull-breadth to width of face (both sexes); that of skull-length to skull-breadth in the case of the males is nearly significant, as is that of skull-length to skull-height in the case of the females. A calculation has been made which shows that the difference of the coefficient of correlation for skull-length to skull-breadth for males and females is

* Printed by the courtesy of the Smithsonian Institution.

entirely due to random sampling. Neither are the differences of the other coefficients of correlation significant in a mathematical sense. It is clear that our samples of males and females are too small to be entirely satisfactory. Though not strictly comparable, it is not

Male

SKULL MEASURE	NUMBER	MEAN	σ	C. OF V.	σ_m		r	σ_r	$\frac{r}{\sigma_r}$
Length	113	184.319	5.9619	3.234	.5609	Length to			
Breadth	113	150.124	5.5878	3.722	.5256	breadth	.25997	.0877	2.964
Ceph. Ind	113	81.549	3.3735	4.137	.3174				
Height	111	131.018	5.1482	3.929	.4886	Length to			
Vert. Ind	111	71.099	3.4622	4.870	.3286	height	.09250	.0941	0.983
Width (Face)	100	142.320	5.3158	3.735	.5316	B. (Skull) to			
Trans. Cran						W (Face)	.53032	.0719	7.370
Facial Ind	100	95.010	3.3511	3.527	.3351				
Upper Fac. H	79	7.770	0.5046		.0569				
Cran. Capac	110	1574.35	105.40		10.05				

Female

SKULL MEASURE	NUMBER	MEAN	σ	C. OF V.	σ_m		r	σ_r	$\frac{r}{\sigma_r}$
Length	73	173.616	6.1123	3.520	.7154	Length to			
Breadth	73	143.137	5.2690	3.681	.6167	breadth	.08560	.1170	0.731
Ceph. Ind	73	82.479	4.0175	4.871	.4702				
Height	73	125.274	4.0755	3.253	.4770	Length to			
Vert. Ind	73	72.315	2.9043	4.016	.3399	height	.29816	.1066	2.805
Width (Face)	67	131.537	4.5657	3.471	.5578	B (Skull) to			
Trans. Cran						W (Face)	.43149	.0994	4.339
Facial Ind	67	91.925	3.5463	3.858	.4333				
Upper Fac. H	65	6.980	0.3668		.0455				
Cran. Capac	74	1405.00	112.09		13.03				

uninteresting in this connection to examine the coefficient of correlation which has been calculated for head-length to head-breadth for Buxton's Mongols (*Am. J. Physical Anthropology*, 11, p. 383), which are males, namely, 0.516 ± 0.102. This is significant. Nevertheless, if we now turn to the corresponding figure for the cranial coefficient, it will be seen (or can be easily calculated) that the difference is entirely due to random sampling.

The figures given above are not strictly comparable with those given by John Cameron (*Am. J. Physical Anthropology*, 11, p. 280 *et seq.*) for the reason that he has selected but 34 cases instead of using all the material available. A calculation has been made which shows that in the case of skull-length (males), though the difference of the averages is quite considerable, nevertheless it is only such as might occur by random sampling. Yet the picture changes decidedly (184.319 mm. as compared with 187); also the skull-breadth (males) is 150.124 mm. as compared with 146. As Mr. Cameron was mainly interested in making some coefficients of correlation of skull-diameters to cranial capacity, it is plausible to think he chose only 34 Mongolian crania as only 34 male and 34 female Greenland Eskimo crania were available for his studies. Even so, the procedure is not justifiable. The entire material should have been used and the standard (or probable) errors given. What I have said as regards Mr. Cameron's figures on Mongolian crania applies to those he gives for the St. Lawrence Eskimo; they are not strictly comparable to those I gave (*Am. J. Physical Anthropology*, 10, p. 489).

The majority of the computations in this paper have been made by my pupil Mr. John Young.

TRUMAN MICHELSON

AN ANATOMICAL EXPEDITION TO AFRICA

An announcement of importance to physical anthropologists, anatomists, and orthopaedic surgeons, has recently been made by President Nicholas Murray Butler, concerning an African Expedition. It has been financed by Columbia University and represents the joint activity of the Department of Anatomy of the College of Physicians and Surgeons (Columbia University) and of the Department of Human and Comparative Anatomy of the American Museum of Natural History.

The chief purposes of the Expedition are, (1) to procure adult specimens of gorillas, chimpanzees, and other African primates, which will be embalmed and prepared in the field for anatomical study in this country; and (2) to obtain motion pictures and other records of postural and foot conditions among the natives for comparative study with corresponding conditions prevailing among peoples subject

to the artificialities imposed by our more highly developed state of civilization.

The enterprise was instigated and planned by Professor Dudley J. Morton of the Department of Anatomy of the College of Physicians and Surgeons, whose work combines physical anthropology with the study of body mechanics as they pertain to orthopaedic surgery. Professor S. R. Detwiler, head of the Department, has most actively supported and helped advance the project throughout its development.

Upon invitation, the American Museum of Natural History entered enthusiastically into the plans and will share in the material obtained. Their cooperation in assigning Mr. Henry C. Raven, an experienced field man and scientist, to serve as leader of the expedition, and Professor William K. Gregory, Curator of the Department of Human and Comparative Anatomy, to collaborate with the University representatives, Professor J. Howard McGregor, of the Department of Zoology, and Professor Earle T. Engle, of the Department of Anatomy (P & S), contributes toward making the expedition one of the outstanding scientific events of the year.

The Advisory Committee comprise the following members: President Nicholas Murray Butler, chairman exofficio; Dr. William Darrach, dean of the College of Physicians and Surgeons; Frank D. Fackenthal, secretary of Columbia; Professor Henry Fairfield Osborn, president of the American Museum of Natural History; and George H. Sherwood, director of the Museum.

The Committee in immediate charge of the Expedition and its related activities consists of Dr. Dudley J. Morton, associate professor of Anatomy (P & S), chairman; the four members forming the personel of the expedition; and Professor Samuel R. Detwiler and Philip E. Smith, of the Department of Anatomy.

The expedition left New York, May 29, on the Aquitania. The African trip will start from the eastern coast at Dar-es-Salaam, proceeding first into the Belgian Congo for specimens and studies of the mountain gorilla (*Gorilla beringei*) in the region of the Parc d'Albert Nationale. From there the journey will move toward one of the tributaries of the Congo River and continue across to the west coast, where in French Territorial Africa, specimens of the lowland gorilla (*Gorilla gorilla*) will be sought. Studies of the natives will be made throughout the trip, and chimpanzees and lower primates collected wherever available. The expedition is expected to return to this country early next year.

Acknowledgment is made of the interested cooperation of Professor John C. Merriam, president of the Carnegie Institute (Washington) and of Mrs. Carl Akeley, in sponsoring the purposes of the expedition, and in contributing commendatory representations to the Belgian and French Governments for necessary permits.

COLLEGE OF PHYSICIANS AND SURGEONS
COLUMBIA UNIVERSITY

QUOTATIONS

DR. JOHN CROSS ON PHYSIOGNOMY AND CONSTITUTION

Nothing is more apparent to the thoughtful student of the history of medical ideas than that the modern study of constitution and personality, in the sense of Kretschmer, Pende, MacAuliffe, Draper, and others, has some of its roots, at least, embedded in such dubious "sciences" as phrenology, physiognomy, palmistry, and the like. In truth, if we leave to one side the obvious quacks who tilled these fields solely for commercial profit, and give attention only to the philosophically minded, it is apparent that the physiognomists, phrenologists, and palmists of the eighteenth and early nineteenth centuries were honestly endeavoring to reach precisely the same result that the constitutionalists of the present day are seeking, namely a sound objective description of the important differentiating criteria or stigmata of the whole personality of an individual. The principal error of the best of these pioneers was simply too far-reaching generalization from too meager and restricted a body of data. But such a procedure is not unknown in other and more respectable branches of science, even at the present time; and by itself would never have brought physiognomy and its neighbors into the state of nearly universal disrepute which they have borne for a couple of centuries. They were ruined primarily by the quacks. In 1741 in England, during the reign of George II, there was passed a statute which denominated as "rogues and vagabonds" all persons "pretending to have skill in physiognomy, palmistry, or like crafty science."

From the point of view of the philosopher, however, there was nothing inherently "crafty" about these "sciences." Thus Dr. Cross, the subject of the present note, defines physiognomy in the following terms:

"To divide and arrange the body into organs, and to ascribe to each its function, is physiology. To view all these organs in connexion, and to compute the influence of each, and the concentrated influence of the whole, in determining the great movements of the individual among other individuals, all acting their respective

parts in the great struggle and bustle of life, is physiognomy. Physiognomy is just a system of corollaries arising out of physiology."

If in this quotation there be substituted for the word "physiognomy" either "constitution" or "personality" it would be difficult to compose a more accurate and satisfactory statement of what the modern effort in this field is concerned with, than is this definition of Dr. Cross published over a century ago.

AN

ATTEMPT

TO ESTABLISH

PHYSIOGNOMY

UPON

SCIENTIFIC PRINCIPLES.

DELIVERED

IN A SERIES OF LECTURES.

BY

JOHN CROSS, M.D.

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1817.

The quotations in this note are taken from a treatise on physiognomy, dedicated to the great Dr. Matthew Baillie, of which the title page is here reproduced in reduced *facsimile*. Dr. John Cross was a Glasgow physician, about whom little seems now to be known. Attempts to locate him in the usual biographical sources failed. I then turned to the never-failing source in medical historical matters, Lt. Col. Fielding H. Garrison, and, as usual, got some real light on the matter, for which I am very grateful. Colonel Garrison writes as follows:

"Your letter opens a very peculiar bibliographical tangle which might be worthy of extended investigation. The book on *Physiognomy* of 1817 is listed in the British Museum Catalogue under John Green Cross and by Callisen under John Cross of Norwich, England; but in his supplementary notes, the old Danish bibliographer corrects himself and makes the *amende honorable* by assigning the item on physiognomy and a book on the *Mechanical Structure of the Human Foot and Leg* published in Glasgow in 1819 to John Cross, M.D., of Glasgow, which is correct. The other man, John Green Crosse

(1790–1850), was a Suffolk man who got his anatomical education at Great Windmill Street and was a friend of Sir Charles Bell, but had no other Scottish affiliations. Some of his books are published under John Cross and some under John Crosse, which is probably the reason for the confusion obtaining in most of the lists under his name. In the Index Catalogue of this Library, the writings of John Green Crosse are all correctly given under his name but we have neither the Glasgow book on physiognomy nor the other one on the foot and leg."

There is so much of extraordinary interest in Dr. Cross' remarkable *Attempt to Establish Physiognomy upon Scientific Principles* that it is difficult to choose for necessarily limited quotation. It has been decided, after careful consideration, to quote two passages somewhat related to each other in their content, in which are stated, in the often flowery and always pious diction of the time, the learned Doctor's views upon the nature of disease, the practise of medicine, the self-regulatory character of the organism as a whole, the selective action of disease in an evolutionary sense—this by the way nearly a half century before the publication of the *Origin of Species*—and the population problem. The first quotation is from pp. 114–117.

"But disease, although it is, on the small scale in relation to man, punishment for human error immediate or remote, yet is, on the large scale in relation to Nature herself, the plan by which she regulates population—that high and important part of her extensive and beautiful economy, by which she balances the sum of life with the means of support. Thus Nature does not inflict punishment for its own sake, but merely administers a little chastisement on individuals, while she is engaged, on the great scale, with arrangements for the happiness and prosperity of the whole.

"The various parts which constitute the living machine are not set, like those of the automaton, upon precarious pivots, nor do the wheels of the animal machine revolve upon their invariable axes with one reciprocity of rotation. There is a considerable latitude given to functions, that they may be able to meet and surmount contingencies; but when the deviations extend beyond the bounds of that wide latitude, then the machinery is driven awry—disease does ensue, of which the very first warning we generally receive is some great struggle of Nature to remedy the evil. Here the duty of the human physician is to keep the whole functions in a condition as near the healthy as he can, and to leave the rest to the Great Physician. Medical men ought, like Hippocrates, to stand by for a little and look on,—first to investigate the disease, and then to inquire whether they should at all interfere. The medical science seems to be fast degenerating into quackery; the grave thoughtful physician has sunk into the busy surgeon with his instruments, and into the smart apothecary with his apron; and operations and doses are, without limit and without discrimination, the ruling order of the present day. The human body is a self-rectifying machine, which requires little more at

our hands than obedience to its dictates, and scope for its own operations. The encouragement given to ignorant drug venders, both licensed and unlicensed, although it does incalculable hurt not only to the deluded dupes, but also to the medical profession, is yet its finest eulogium. That medicine might not interrupt the final cause of disease, medicines are constituted poisonous. How nice and how difficult then must the medical profession be, when we are thus necessitated to expel one poison out of the body by the introduction of another? In order to understand pathology aright, we must first make ourselves masters of physiology; for the real cause of so much absurdity in medical theories has been ignorance of physiology. Before we can understand the nature of disease, we must first understand the nature of health, from which disease is a deviation. We must not only attend to all the functions individually, but must also examine and scrutinize the relations that subsist between them, until we come to see all the various organs of the body go to constitute one single great organ. Then will we rightly understand the mechanism of the human body, and be able to say where, and how, and why, the machinery has gone wrong, and what shall put it right, before we think of lifting a tool. Then, instead of prescribing the antispasmodic for the spasm, the anodyne for the pain, and for every occurring symptom its respective antidote, the guardian of health, like the guardians of life and property, instead of thus stopping, with a palliative, the mouth of every crying symptom, summons them all before him, deliberately hears their evidence, and rids them of the lurking culprit."

Could he but know it Dr. Cross would no doubt be enormously pleased to find that the development of modern diagnosis and treatment has been so precisely along the lines outlined in his prophecy!

The second quotation is from pp. 128–131.

"It is a maxim in political economy, that no more people can live in the world, or in any part of it, than there is food to support. In rude states of society, redundant population is swept off by actual starvation. Here hunger is the great stimulus to exertion. But in the more advanced stages of society, animal exertion and improvement are taken out of the charge of the gross appetite, and transferred to more intellectual incentives. Here, therefore, needless starvation is precluded, and the superfluous visitors are politely dismissed, without being told that there is no food for them. The Great Physician of nature takes care, in the first place, that the disease of over-population shall not spread. This is immediately effected by the restraints which arise to marriage. A dose of contagious epidemic is then administered, which, passing through all the tortuous windings of life, purges off the refuse; for where no interference is given, a contagious disease carries off the weak and the decrepid, while the robust and shapely pass through more purified than before. In the performance of this gloomy but necessary work, glimpses of divine benevolence may be discerned, not only in the ultimate design of the work, but also in the mode of the performance. Contagious diseases are febrile; in other words, have their primary seat in the brain; so that the patients are comparatively insensible of their misery. Had contagious diseases carried off all indiscriminately, old as well as young, the

parent as readily as the child, to what sum might not human misery have amounted? The death of a child is only the removal of an unproductive eater, but the death of parents is not only a loss of useful members, but also throws a burthen of unproductive eaters on the back of society. Nature therefore has so ordered matters, that contagious diseases shall attack and carry off the young almost exclusively. For this purpose these diseases are allowed to attack a person only once in a life-time; so that parents have for the most part undergone a prophylactic. Moreover, these diseases are inflammatory, to which kind of disease the vascular bodies of children are peculiarly conducive. That contagious disease shall most certainly and effectually accomplish its purpose of thinning society, respiration itself is the medium of communication, and thus the blow is struck at the very centre of life. This kind of disease seems to be generated by too much respiration for a length of time in a given quantity of air. Here nature does not wait till the whole atmosphere shall be polluted; but, whenever too much breathing takes place in any part of the atmosphere, then she infers that over-population is general, and nips the evil in the bud, by sending forth a contagious epidemic. By ordaining that too much breathing for a length of time in a given volume of air shall generate contagion, nature shows her aversion to too much crowding together of her creatures; and, in the strongest of language, commands them to spread over the surface of that earth which was given them for cultivation. By obeying this order, two grand ends are gained at once, either of which, without the other, must always be insufficient for human welfare,—an increase of food and population, and an atmosphere fit, in all its extent, for respiration."

R. P.

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Institute for Biological Research, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

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